BRAIN:

A JOURNAL OF NEUROLOGY.

VOL. X.

BRAIN:

A JOURNAL OF NEUROLOGY.

EDITED

FOR THE NEUROLOGICAL SOCIETY OF LONDON,

BY

A. DE WATTEVILLE.

VOL. X.

London:
MACMILLAN AND CO.
AND NEW YORK.
1888.

LONDON:
PRINTED BY WILLIAM CLOWES AND SONS, LIMITED,
STAMFORD STREET AND CHARING CROSS.

LIST OF CONTRIBUTORS.

ANDERSON, JAMES, M.D., F.R.C.P.
BANHAM, FRENCH, M.A.
BASTIAN, H. CHARLTON, M.D., F.R.S.
BATTERHAM, J. W., M.B. (LOND.), F.R.C.S.
BENNETT, A. HUGHES, M.D., F.R.C.P.
BIRT, ERNEST.
BRAMWELL BYROM, M.D., F.R.C.P. (EDIN.).
BRISTOWE, J. S., M.D., F.R.S.
BRUCE, J. MITCHELL, M.D., F.R.C.P.
BULLEN, F. ST. JOHN.
BUZZARD, THOMAS, M.D.
CARTER, ROBERT BRUDENELL.
CRICHTON - BROWNE, SIR JAMES, M.D., F.R.S.
DALY, EDWARD O., M.A., M.D. (OXON.), M.R.C.P.
DONKIN, H. B., M.B. (OXON.), F.R.C.P.
DRESCHFELD, J., M.D., F.R.C.P.
DRUMMOND, DAVID, M.D.
FERRIER, D., M.D., F.R.S.
GAY, WILLIAM, M.D., M.R.C.P.
GREENLEES, T. DUNCAN, M.B. (EDIN.).
HAYCRAFT, J. B., M.B., B.Sc.
HILL, ALEX., M.A., M.D.
HORSLEY, VICTOR, B.S., F.R.S.
HUGHLINGS-JACKSON, J., M.D., F.R.S.
HUTCHINSON, JONATHAN, F.R.S., LL.D.
IDELSON, VALERIUS, M.D.
MAGUIRE, ROBERT, M.D.
MERCIER, CHARLES, M.B.
MOTT, FREDERICK W., M.D., M.R.C.P.
MYERS, A. T., M.D.
OLIVER, JAMES, M.B., F.R.S.
ORMEROD, J. A., M.D., F.R.C.P.
PIERSON, R. H., M.D.
RABAGLIATI, A., M.D.
ROSS, JAMES, M.D., LL.D., F.R.C.P.
SAUNDBY, ROBERT, M.D. (EDIN.); F.R.C.P. (LOND.).
SCHÄFER, E. A., F.R.S.
SHARKEY, SEYMOUR J., M.B., F.R.C.P.
SUCKLING, C. W., M.D. (LOND.), M.R.C.P.
SULLY, JAMES, M.A.
SUTTON, J. BLAND, F.R.C.S.
THORBURN, WILLIAM, M.D., B.S., B.Sc. (LOND.), F.R.C.S.
TOOTH, HOWARD H., M.B.
TUKE, D. HACK, M.A., M.D.
WATTEVILLE, A. DE, M.A., M.D., B.Sc.
WHITE, W. HALE, M.D.
VOORTHUIS, J. A., M.D.

LIST OF ILLUSTRATIONS.

ERRATUM.

On page 234 of the present Volume, the last paragraph of Case I., beginning with the words, "This gland," etc., is the concluding paragraph of Case II., and has been accidentally transposed.

CONTENTS.

ORIGINAL ARTICLES:—

CLINICAL CASES:—

CRITICAL DIGESTS :—

REVIEWS AND NOTICES OF BOOKS :—

ABSTRACTS OF BRITISH AND FOREIGN JOURNALS :—

BRAIN.

APRIL, 1887.

Original Article.

THE "MUSCULAR SENSE"; ITS NATURE AND CORTICAL LOCALISATION.[1]

BY H. CHARLTON BASTIAN, M.D., F.R.S.

Professor of Clinical Medicine and of Pathological Anatomy in University College, London.

HAVING been honoured by a request from the Council of the Neurological Society to open a debate upon "The Muscular Sense; its Nature and Cortical Localisation," I willingly undertook this duty, not because I thought the task an easy one, but, recognising to the full all the inherent difficulties of the subject and the unsettled state of opinion thereon, I fully concurred in the notion that such a discussion might do much good. If it does not lead to the immediate settlement of the many points still in dispute, it may, at least, serve to bring out into clearer light the nature of the problems to be settled by future workers and thinkers, in order that unanimity may ultimately prevail. I say "thinkers" advisedly, because this is eminently one of those subjects on which observation and experiment alone will not suffice, especially observations and experiments conducted upon lower animals.

INTRODUCTION.

It may be regarded as a physiological axiom, that all purposive movements of animals are guided by sensations or by afferent impressions of some kind.

Physiologists know that the several kinds of movements

[1] Paper read before the Neurological Society of London, on December 16, 1886.

which animals are accustomed to perform are only artificially and for purposes of convenience divided into such classes as (1) reflex or automatic, (2) secondary-automatic, (3) instinctive, (4) ideo-motor, or (5) volitional.

From the point of view of the dependence of these several kinds of movement upon sensation, or upon afferent impressions either actual or revived, no fundamental distinction can be drawn between them.

In reply to this it might be alleged that volitional movements constitute a class apart, seeing that their performance implies as a necessary factor, which the others do not, the presence of, and illumination derived from, Consciousness. But it must be borne in mind, (*a*) that Consciousness is not itself a factor, it is rather of the nature of an epiphenomenon; (*b*) that it exists in varying degrees during the executions of different kinds of voluntary movements, being of maximum intensity during the performance of unfamiliar muscular actions and of minimum intensity (almost vanishing) in association with easy and familiar voluntary acts; and (*c*) that as a matter of fact, it is often almost impossible to draw anything like a dividing line between ideo-motor and voluntary movements, so insensible are the gradations between them.

If we compare for a moment a simple reflex movement with a volitional movement, some important differences may nevertheless be found, which are of great significance from the point of view of the subject with which we are now concerned.

A reflex movement may be evoked and guided to its completion by afferent impressions which are wholly unfelt; we have here apparently nothing else than a direct relation established between the paths of afferent impressions and the nervous cell and fibre tracts needful for evoking the suitable activity of some definite sets of muscles. In other words, the line of least resistance for the molecular movements set up in the cells on the afferent side is found to lie along the intermincial fibres connecting these with certain groups of motor cells, and thence outwards through their efferent nerve fibres to the muscles. Even in ideo-motor movements something of the same kind seems to occur; that is to say, there seems to be an equally well-formed beaten tract (by means of nerve fibres) between the cells of the perceptive centres in which

the idea or revived sensory process occurs, and the particular motor mechanisms connected therewith, whose excitation gives rise to the fitting movement.

But simple, familiar, voluntary movements are attended by so little conscious illumination as to be often indistinguishable in every way from ideo-motor movements. The utterance of words in ordinary speech, that is, in the person's own language, is effected by movements which may be described either as voluntary or as ideo-motor. The whole act of speech is truly a voluntary performance; but the enunciation of each word takes place with all the ease and lack of attention typical of an ideo-motor movement. It is, however, in the performance of a new or unfamiliar voluntary action that we find the widest divergence from the phenomena attendant upon the execution of a simple reflex movement. Here we have the full blaze of consciousness, the whole attention of the performer, directed to the production of the movement in the desired manner, though this result may not be attained till more or less marked failure has occurred on many occasions. What can guide these endeavours to perform new movements save sensory impressions, actual or revived? What is the result of this concentration of attention upon present and revived sensory processes? This latter question cannot be answered definitely and in detail, but seeing that one result of practice is to make the new movement after a time quite easy of execution, and that further practice still causes it to recur with such facility that it may be fully entitled to take rank as an 'ideo-motor' or even as a 'secondary automatic' movement, we are fairly entitled to conclude, that one of the hidden effects whose existence we can only infer has been the laying down of beaten paths for efferent stimuli, from the sensory centres which have been concerned in guidance (the stimuli in question being in part, at least, the molecular movements occasioned by the revived activity of the sensorial centres) to the particular combinations of motor nerve mechanisms whose excitation is needful for evoking the movements in question.

But if it be assumed that this is what occurs when, by dint of long practice, an at first difficultly-executed voluntary movement becomes, in process of time, a movement so easy of execution as to recur independently of conscious attention,

with machine-like regularity, three important consequences follow:—

In the first place, it seems to afford an organic proof that even as in ideo-motor actions, so in voluntary movements, the immediately evoking stimuli flow out from the sensory centres concerned with the production of such movements—the voluntary movement grows into the ideo-motor movement, and then unmistakeably the guidance is wholly through the sensory centres, for then (as we have seen in the case of a simple reflex movement) the line of least resistance for molecular movements from the sensory cells is through certain new communicating fibres bringing them into relation with definite groups of motor nerve mechanisms.

Secondly, it seems clear that the same motor mechanisms must be concerned with the production of the new voluntary movements as with the ideo-motor or secondary automatic movements into which they become converted at a later date. We have to do in these two sets of cases with differences in the degree of perfection of the nervous mechanisms, and with different degrees of perfection in the functional and structural relations between the sensory and the motor mechanisms especially, rather than with an alteration in the position in which the movements are, so to speak, organised. It is, therefore, in my opinion, a fundamental error to look for special voluntary motor centres The strictly motor side of voluntary motor mechanisms should be identical with, or lie side by side with, the strictly motor side of "ideo-motor" or "secondary-automatic" mechanisms of the same type.

Thirdly, we may safely conclude, that when once the ways or beaten tracts have been laid down by which certain sensory departments are connected with certain motor mechanisms for the performance of voluntary movements, the former may be incited to renewed (or ideal) activity, and may be followed by appropriate responsive movements, almost if not quite independently of, and certainly without any need for, Consciousness as an attendant of such revived sensorial activity: in other words, when once the internuncial tracts have been clearly established from cortical sensory centres to motor mechanisms, it is not in the least necessary that Consciousness should be an appanage of this revival, since even the unfelt recall by associa-

tional processes of activity in the guiding sensory centres, may by causing an outflow of molecular movements, now suffice with machine-like regularity to evoke the accustomed motor reactions, as in the case of a skilled dancer or a skilled player upon a musical instrument.

SENSATIONS RESULTING FROM MOVEMENT: KINÆSTHETIC IMPRESSIONS.

Before entering into any details concerning the sensory guidance of movements in general, something requires to be said about one particular class of sensory impressions which are of great importance in this relation. I refer to the body of sensations which result from or are directly occasioned by movements. This constitutes a complex of impressions which, for the sake of convenience of reference as well as for the purpose of indicating their common functional relations, I have proposed[1] to include under the designation *Kinæsthesis*, or the "sense of movement," perhaps I ought rather to have spoken of "sensations of movement," as Wundt does,[2] but the former term is more in accordance with those already in use in connection with this subject (e.g., "muscular sense," and "sense of force"). Impressions of various kinds combine for the perfection of this "sense of movement," and in part its cerebral seat or area corresponds with the sense of touch. Thus, under it are included, as its several components, cutaneous impressions, impressions from muscles and other deep textures of the limbs (such as fasciæ, tendons, and articular surfaces) all of which yield conscious impressions of various degrees of definiteness; whilst, in addition, there seems to be a highly important set of unfelt or but little felt impressions which guide the volitional activity of the brain, in ways hereafter to be defined, and which serve to bring it into relation with the different degrees of contraction of all muscles that may be called into action.[3]

[1] 'The Brain as an Organ of Mind,' 1880, p. 543.

[2] Wundt says:—"Les sensations du mouvement, comme nous avons montré précédemment, sont pour nous *des produits fusionnés complexes*, provenant de sensations d'origine différente" ('Élem. de Psych. physiol.' Trad. franç., 1886, i., p. 421.

[3] I have introduced a qualifying phrase after the word "unfelt" above, because

By means of this complex of sensory impressions we are made acquainted with the position and movements of our limbs, we are enabled to discriminate between different degrees of "resistance" and "weight," and by means of it the brain also derives much unconscious guidance in the performance of movements generally.[1]

Of course it may be easily said, and doubtless will be said, by those who have not yet realized the importance of bestowing special attention upon this complex of impressions, that it is unsuitable to describe as though it were a distinct endowment, the means by which we receive and appreciate a group of impressions having such diverse sources of origin. This mere technical or formal objection seems to me, however, to have little weight, when we consider that from a functional point of view they constitute a group of impressions altogether apart, recurring with extreme frequency, and always in more or less similar combinations, during almost every hour of our waking life—subserving also, as we hope hereafter to show, a most important purpose. I think, therefore, we are warranted in speaking of the reception of this group of sensations under the name of kinæsthesis, or the sense of movement. There will certainly be a real convenience in being able to allude to such impressions briefly, as 'kinæsthetic impressions.' It is confessedly a mixed group partly "intrinsic" and partly "extrinsic" in their origin.

---

on reflection I think I have been wrong hitherto in saying that "muscular sense" impressions are wholly unfelt. There must be a kind of consciousness associated with them if, as seems now to be established, our knowledge of the position of our limbs, as well as of differences in weight, is in the main due to "muscular sense" impressions.

[1] These impressions are almost always closely linked with others of a tactile, visual, or auditory order, so that their associated ideal recall presents no sort of difficulty, and the question whether, when recalled, they reveal themselves in consciousness or not is one which has no sort of importance for, as Ribot says, consciousness "n'est cause de rien." Writing in the 'Revue Philosophique,' June, 1884, p. 639, L. Manouvrier pointedly illustrates this in the following manner: "En son absence, les cellules cérébrales peuvent être le théâtre d'opérations très parfaites auxquelles peuvent succéder des mouvements très bien appropriés et coordonnés, tout comme s'il y avait eu production de conscience. La seule différence est que le cerveau sert alors de substratum à ces opérations nerveuses sans le connaître, tandis que, dans l'état opposé, il les subit consciemment, voilà tout."

From one point of view, especially, important differences separate this synchronous and fused group of impressions from all other sensory impressions—differences to which it will be as well at once to allude.

Ordinary extrinsic sensorial impressions commonly play the part of instigators to movement. Thus impressions of smell, sight, hearing, touch, and taste are either the immediate or the remote instigators of the great majority of the movements executed by animals.

The movements so stimulated are in almost all cases of a more or less purposive type, directed, that is, to the attainment of some end.

During the whole time that such movements are being evoked the animal is constantly receiving those groups of mixed afferent impressions which we have styled "kinæsthetic," called into existence by the mere movements themselves.

Kinæsthetic impressions are never instigators of movement in the same sense that olfactory impressions may be instigators of movement. Visual impressions and auditory impressions are not only frequent instigators of movement, they are also respectively all-important guides for certain classes of movements. From a functional point of view, kinæsthetic impressions are guides only; these functional uses are, however, co-extensive with movements themselves, since in conjunction either with visual or with auditory impressions they act as guides for all sorts of movements. As it is with visual and auditory, so is it with kinæsthetic impressions, their guiding influence in the production of movements is brought to bear partly under the form of actual sensations and partly under the form of revived impressions (the memories of past activity).

Seeing that kinæsthetic impressions must be as numerous and as endlessly diversified, in their kinds and combinations as movements themselves, it follows that large tracts of the brain ought to be concerned with their registration for future use in the guidance of all kinds of voluntary movements.[1]

[1] I purposely leave out of account for the present the fact, that kinæsthetic impressions enter largely into the composition of nearly all our visual and tactile impressions; movements of the eyes, or movements of the hands and fingers, not only vastly increasing the range of possible visual or tactile impressions, but also

Except during the last forty years, the occasional references which have from time to time been made to kinæsthetic impressions have appeared rather in the writings of philosophers, than in those either of physiologists or pathologists. Yet the first mention of such impressions goes back, according to Sir William Hamilton, to a rather remote past. He tells us that two Italian physicians, Julius Cæsar Scaliger, in 1557, and Cæsalpinus of Arezzo in 1569, quite independently of one another, were the first to recognize and definitely state that the exercise of our power of movement is the means whereby we are enabled to estimate degrees of "resistance," and that by a faculty of "active apprehension" which was by them contrasted with touch as "a capacity of sensation or mere consciousness of passion." These early references to such an endowment, like those of Maine de Biran and Sir William Hamilton himself, have for the most part supposed the existence of a separate faculty or endowment associated with volition, or the mere will to move, to which such names as the "sense of effort," the "sense of force" or the "locomotive faculty" have been given—agreeing very closely with what Wundt has since termed the "sense of innervation."

Some of the causes why physiologists should for so long have paid but scant attention to this class of impressions may be thus enumerated :—

(1) The most typical and important group of kinæsthetic impressions, viz., those derived from the muscles themselves, belong like visceral impressions, to the class of "intrinsic" sensations, which, as a whole, receive but little of our conscious attention under all ordinary conditions of life. These are "means" rather than "ends," and consequently soon become sub-conscious modes of activity.

(2) These impressions are not directly concerned with the organism's life of relation—that is, they do not directly

---

entering as it were into their very structure, and thereby enabling us to recognise and judge what philosophers call the primary qualities of matter, viz., solidity, extension, figure, etc. I also omit further mention of the fact, that kinæsthetic impressions likewise act as guides in the great class of reflex movements which are produced independently of the cerebral hemispheres, by the mesencephalon and cerebellum, as well as by other sensori-motor couples of lower grade.

stimulate its converse with the outside world. Impressions derived from movement cannot in themselves be, nor are they when ideally revived, first-hand instigators of movement, however much they may aid in the repetition of movements previously effected.

(3) Owing to the nature of this sensory endowment, it is one which cannot be investigated by experiments upon animals. The presence or absence of ordinary sensory endowments may be tested in the lower animals, by looking for the recurrence or not of some particular motor reactions which we know are apt to be excited by the presentation of this or that stimulus to the sense which is being tested. The sense of movement being however a so-called "intrinsic" sense, and not an instigator of movements, cannot be tested in this manner. Indeed it may be safely said that no experiments that can be made upon the lower animals, even upon monkeys, are capable of throwing any decisive and direct light upon the presence or absence of the most important element in this endowment, viz., the impressions emanating from contracting muscles.[1]

The Muscular Sense.

It should be stated here that the announcement of the existence of a "special class" of sensations emanating from muscles, came from Sir Charles Bell. He first postulated the existence of a special endowment, to which he applied the name "*muscular sense*," whilst the reality of the sensations emanating from muscles was first demonstrated and accurately estimated by the experiments of E. H. Weber,[2] who, however,

[1] Thus it happens, perhaps, that the evidence on which Ferrier relies in support of the notion, that the "muscular sense" was defective in three monkeys operated upon by him (*See* 'Functions of the Brain,' 2nd ed., 1886, pp. 335, 340, 344), is no more convincing to me, than is the evidence of Hitzig and Nothnagel to him, in support of their cortical localisation of muscular sense centres (p. 379). We must have observations on men and women for the solution of this problem, who can *tell* us what they know of the position of their limbs, and of their movements (active or passive), as well as concerning their ability to appreciate differences in weight.

[2] Annotationes anatom. (Progr. collecta.) Proleg. xii. (1831). *Tastsinn und Gemeingefühl.*

spoke of the endowment by which we are enabled to discriminate different degrees of "resistance" or weight, as the "sense of force." E. H. Weber was the first to make definite investigations as to the differences in weight which could be appreciated by the mere tactile sense of contact and pressure whilst the hand was at rest on a flat surface, and, subsequently, to ascertain by careful experiments that a considerably heightened power of discrimination existed when the muscles were called into play—that is, by no longer allowing the hand to rest upon a flat surface, but by slowly moving the arm when different weights were successively placed upon the palm. Numerous comparative experiments of this kind made by Weber, and since confirmed by other investigators, have shown, that the activity of the muscles adds greatly to our ability to discriminate differences in "weight" or "resistance." Thus, by the mere sense of cutaneous pressure it has been found that the addition of one-third of the original weight, whatever it may have been, is needful to produce a perceptibly different impression; while, on the other hand, when muscles are allowed to be called into play the power of discrimination is so greatly heightened that an addition of no more than $\frac{1}{17}$th of the original weight is generally capable of yielding a distinguishable difference in the resulting impressions.

This constitutes, in fact, the physiological proof of the existence in us of a distinct ability to discriminate different degrees of weight or resistance by impressions resulting from various states of tension or contraction in our muscles. This ability is lost in certain diseases, and its absence gives rise to disordered movements when the eyes are closed, as well as to ignorance of the position of the limbs and other related defects.

For the better understanding of this part of our subject it seems best to record here, in sequence, the different cases to which I desire to call attention, for the purpose of illustrating the nature of the defects themselves, as well as the nature of the diseases with which they are most commonly associated.

As samples of this kind of evidence I will first cite a few details (all that I have been able to find) concerning two cases which were reported at a time when no particular attention had been given to such morbid phenomena.

"In February, 1790, a German schoolmaster, after extreme worry, was seized with an insensibility extending over the whole body. Movements were freely executed at all the joints, and all his muscles were entirely under the influence of the will, though the patient had no feeling of having executed movements. He could move his toes separately or together at will, but in order to know whether the movement had really taken place he was obliged to assure himself by means of his eyes. He walked quite easily, though it seemed to him as if he did not make use of his own legs."[1]

Demeaux[2] gives details concerning a woman suffering from hemi-anæsthesia, which show the complete loss in her of the sensations coming from muscles, as well as of all other modes of sensibility, superficial and deep, in the affected limbs. He says:—"She put her muscles in action under the influence of her will, but she had no consciousness of the movements which she executed; she knew not what was the position of her arm—it was impossible for her to say whether it was extended or flexed. If one told the patient to raise her hand to her ear, she executed the movement immediately; but when my hand was interposed between her own and the ear, she was not conscious of it; if I stopped her arm in the midst of its movement, she did not become aware of it. If I fixed, without allowing her to be aware of it, her arm upon the bed and told her to raise the hand to her head, she strove for an instant and then became quiet, believing that she had executed the movement. If I induced her to try again, showing her that her arm had remained in the same place, she attempted to do so with more energy, and as soon as she was compelled to call into play the muscles of the opposite side [of the body], she recognized that the movement was opposed."

In these cases the so-called "muscular sense" was clearly absent, and so were all kinæsthetic impressions in the affected limbs, leading to an ignorance of their position and movements. Details will now be given concerning another very remarkable case, of a somewhat different type, which was examined with great care and reported at length by Landry,[3] in his interesting "Mémoire sur la paralysie du sentiment d'activité musculaire."

The patient was a physician, fifty-five years of age, who, from Landry's account, I judge to have been clearly suffering from

[1] Reported in Hufeland's 'Journal,' and quoted by Bellion in his 'Recherch. sur la Pathol. et Physiol. des Sens. tactiles,' Thèse de Paris, 1853, p. 46.

[2] 'Des Hernies Crurales,' Thèse de Paris, 1843, p. 100.

[3] 'Gaz. des Hôpitaux,' 1855, Obs. III., p. 270.

locomotor ataxy of an unusual type. In regard to his power of executing movements with his eyes open and closed respectively, and as to his knowledge of his movements and of the different degrees of tension in his muscles, the following quotations may be made:—

"Voluntary contraction seemed to have lost nothing of its energy. When seated or in the lying posture he easily elevates his lower limb and maintains it elevated for a long time without apparent fatigue, only the limb is the seat of oscillations which he always in part controls when he sees them. He has no idea of the more or less extensive and violent passive movements which his limbs are made to undergo. Active movements are not even appreciated as soon as he ceases to watch them. Thus, after having made him close his eyes, if he is asked to move one of his lower limbs in whole or in part, he does it, but cannot say whether the movement executed is great or small, energetic or feeble, or even whether it has really taken place; and when made to open his eyes whilst he is moving his leg from right to left, for example, he declares that he had only a very inexact idea of this action. In the same way if after having specified to him some aim to be accomplished, and having made him measure the distance with his eye, he is told, without looking, to bring his foot to the spot, it remains on this side of it or goes beyond without his suspecting it, or if he succeeds it is only by chance. On the contrary, when he watches the movement of his foot he easily places it correctly. . . . In fact, when he cannot see his limbs, he is no longer conscious of their existence—he is ignorant of their position. When in his bed he loses them, so to speak, and has to search for them with his hands. In the sitting posture, he often seeks to extend or to flex them when they are already in these positions. If, having the wish to execute a movement, I prevent it, he does not perceive it and attributes to the limb the position which he had intended to give to it. He neither appreciates the resistance one opposes to his movements, nor weights with which one charges his limbs. He easily raises a very heavy object when he has estimated its weight by sight; but if he is not permitted to judge of this previously, he does not know how to apportion his effort to its weight, and the muscular contraction is either too great or decidedly too little.

"In the upper limbs there are almost the same disorders: ill-co-ordinated and badly measured movements; defective appreciation of passive movements, and even of active movements when the patient does not see them; but faculty of co-ordinating and of measuring his movements when he calls to his aid his sense of

sight; great energy of muscular contraction; integrity and rapidity of each isolated movement of the fingers, of the hands, of the fore-arms, and of the arms; impossibility of appreciating weight or resistance; sensation of extreme lightness of the hands, etc. Prehension is much altered; the patient seizes an object clumsily, and only succeeds in seizing it better when he attentively watches his fingers. He can no longer write. As soon as his attention is relaxed he lets objects fall which he may have been holding, without perceiving that their weight has ceased, or that his fingers are extended. Everything appears to him without weight. He can neither appreciate the thickness nor the size, nor the form of bodies which one makes him touch, or rather almost always attributes to them a form and a size almost identical—he describes them as cylindrical or round, and of about two centimètres in thickness. If, after having made him close his eyes, one takes from his hand an object which he is holding, he does not perceive it, and continues to maintain the position he had taken in order to hold it, believing that he feels something of the same shape and volume. The same thing happens if, equally without his knowledge, one has completely opened his hand; he believes it to be still closed and filled by an object whose form and size he judges as if he had never ceased to hold it. He does not perceive the resistance which is opposed to the movements of his fingers and hand, and often thinks that he has executed a movement when he has been prevented from doing so. He cannot judge of the consistence of bodies, to which he attributes without discernment either hardness or softness. When one plunges, without his knowledge, one of his fingers in water, he perceives the coldness very well, but when he is asked to move his finger about, he thinks he moves it in air and does not perceive the resistance of the liquid.

"His movements have, as I have said, a great irregularity; but all, even the most delicate movements of the fingers, are executed with the greatest facility. When he looks at them one can only prevent their flexion with difficulty, and he can press one's hand vigorously. If he ceases to see, he continues to make efforts to flex his finger or his hand when opposed, but he does not know how to apportion his contraction to the resistance; he also presses the hand very softly, as if there were a real loss of power. With his eyes open, he easily opposes the thumb to each of the other fingers; with his eyes closed, the movement of opposition occurs, but the thumb only by chance meets the finger which it seeks. With his eyes open he is able, without hesitation, to bring his two hands together; but when his eyes are closed his hands seek one another in space, and only meet one another by chance.

The fingers can no longer be regularly interlocked. There is not the least tremor of the hand."

"*Condition of cutaneous sensibility in the four limbs.*—Sensations of contact preserved but obtuse; M. X. feels quite well when he is touched, but he cannot say with what kind of object. Thus he cannot decide whether it is with wool, with wood, or with the observer's hand. He no longer feels tickling. He is able perfectly to specify the precise point at which he is touched, unless the body with which the contact is made covers only a very small area of skin, as, for example, the blunt point of a needle. Sensations of temperature intact; sensations of pain much exaggerated."

Landry gives two other cases very similar to the above, but I will next quote a few details concerning a very remarkable case which, in one important respect, stands in marked contrast with those that have gone before, and more especially with the two cases which will follow—I mean, as regards the patient's ability to execute precise movements when the eyes were closed. This is a case in which a more or less general anæsthesia existed for over thirty years, and the man was repeatedly examined by many trustworthy observers. The record of the case was published by Spaeth,[1] and the autopsy was fully described by Schueppel.[2]

Remigius Lens, aged forty-two, in the year 1862, has suffered for twenty years with anæsthesia of the hands and arms, which rapidly became severe; for six years similar troubles have existed in the lower extremities. Present condition: upper extremities wholly anæsthetic; on the soles of the feet the sensations of touch, pressure, and pain are entirely extinct, and in the legs considerably diminished. *He falls when his eyes are closed.* In the dark, when in bed, he feels as if floating in the air, as the anæsthesia extends to the trunk.

March, 1864.—Sense of pressure in the upper extremity, and the sense of force, entirely extinct. Sense of position of the upper extremity and of passive movements of the latter completely extinct. Movements of the upper extremity powerful and perfectly correct; the patient eats alone, dresses himself, etc., as far as he can direct his acts with his sight. When the eyes are closed, the hands are moved nearly like those of a blind man. In the lower extremities, besides the cutaneous anæsthesia, there is

[1] 'Beitr. zur Lehre von der Tabes dorsalis,' Tübingen, 1864.

[2] 'Arch. d. Heilk.,' Bd. xv., 1874, p. 44.

complete loss of the sense of passive movements and of the position of limbs. In spite of this the patient can walk without support, quite fast and securely, for a good distance. If he is asked to raise his foot to a given height while his eyes are shut, he accomplishes the act by a perfectly quiet and suitable motion.

June, 1872.—Sensibility continues the same. When the eyes are shut he has no idea at all of the position of his limbs, and, if standing falls. He can still walk clumsily, but not atactically. He can perform all desired actions with his arms, as long as he can see them.[1]

This patient seems to have had decidedly more power of executing accurate movements when the eyes were closed, than is commonly met with in such cases. But then the anæsthetic condition of the arms had lasted for twenty to thirty years when the results above recorded were obtained, so that ample time had elapsed to permit his nervous system to accommodate itself to its altered conditions, and so to gain a power of executing voluntary movements with closed eyes, which probably did not exist at first in this patient, in whom "muscular sense" impressions appeared not to reach the cerebral cortex, however it may have been with lower centres. This is certainly an altogether exceptional case.[2]

Duchenne's attention seems to have been attracted to the effects of loss of the "muscular sense" very shortly after the first cases were published by Landry.[3] A short paper from him, entitled "Usages de la sensibilité musculaire" appeared in 1853,[4] which two years afterwards was reprinted and added to in his work "De l'électrisation localisée" (p. 410). Here Duchenne points out that such cases as I have above referred

[1] Abstract quoted from translation of Ziemssen's 'Cyclopædia,' vol. xiii., p. 89.

[2] Yet we find Dr. Ferrier relying upon it, in spite of all other evidence to the contrary, apparently as sole support of a view, which will assuredly find no general acceptance, to the effect that a "limb can be moved freely and forcibly and directed without uncertainty, for volitional purposes," when the eyes are closed, "in the entire absence of any sense of movement" (*loc. cit.*, p. 65). In regard to that point I would refer him to the cases of Landry, as well as to those now about to be recorded from the observations of Duchenne, Briquet and Bazire (pp. 16–20).

[3] Archiv. génér. de Méd., 1852. Other cases were recorded in 1853 by Bellion (Thèse de Paris) 'Rech. histor. sur la pathol. et la physiol. des sens. tactiles.

[4] 'Moniteur des Hopitaux'

to (excepting the last, which, of course, appeared after the date of his work) constitute only one category of those in which there is paralysis of what Landry termed "le sentiment d'activité musculaire." He says:—"In the second category, which is much less numerous, *the patients when similarly deprived of sight lose the faculty of executing the simplest voluntary movement.* If they are bid for example, to open or shut the hand, flex or extend the forearm, in a word, whatever movement one asks them to make, the muscles which ought to enter into contraction remain inert, notwithstanding all the efforts of the will. One only observes sometimes certain irregular movements, slight in extent, weak, and different in kind from those that they wish to execute—movements of which they have even no knowledge. Nothing can describe their astonishment when they perceive, after the experiment, that their limb has remained in the same situation, when they thought they had made it execute a movement. Their surprise is all the more great, since they can execute this same movement with rapidity the instant that they are permitted to look at the limb."

In confirmation of these statements Duchenne gives a detailed account of a patient in 'la Charité' under the care of Briquet, whom they conjointly submitted to repeated careful examinations.[1] The patient was an hysterical girl who was suffering from complete anæsthesia, superficial and deep. She presented all the same kind of phenomena as were seen in the cases already recorded, that is to say a lack of knowledge as to whether a movement which had been willed had been executed or not (necessarily, therefore, an ignorance of the extent of her movements and of the position of her limbs), together with a total inability to appreciate degrees of resistance. In addition, however, there were new peculiarities concerning which the following quotations may be made.

"There was also another phenomenon which M. Briquet and I observed in this case of general and profound insensibility: that is, one could violently strike her limbs, shake them violently, and change their place, without her having any knowledge of it. . . .

[1] *Loc. cit.* p. 417.

On devoting myself to the examination of this patient I found that she presented in the highest degree the phenomena which constitute the special object of this article. Thus having stooped in such a manner as to prevent her seeing her hand at the moment when I told her to close it, the hand remained motionless though she thought she had closed it; and, whilst I was making her extend and flex the forearm upon the arm, having turned her attention away from this limb the movement was immediately arrested—the limb remaining in position as if it had been tetanised. I should add that the patient, whom I had seen for the first time, was quite ignorant of what I sought to establish. . . . But here is another experiment which shows, in addition, that the action of the will in combination with the sense of sight was necessary also for the cessation of a contraction once produced. If after having made her squeeze the hand, she is prevented from seeing it, and told to cease all effort, one feels that the contraction of the flexors continues, and it is necessary to employ considerable force to open her hand. Or, if after having made her flex the forearm, one prevented her seeing it the forearm remained in a state of flexion, and it was necessary to employ a pretty strong force to extend it (it must be recollected that the patient, deprived of all sensibility, had no consciousness of the movements which were impressed upon her limbs").

"Here, again, is another experiment which shows that it is not sufficient for the patient merely to be able to see in order that the movement should be obtained, but that it is also necessary for her attention to be fixed upon the limb about to be called into movement. Having placed the patient's hands sufficiently close to one another so that she was able to see them both equally well, I asked her to shut them and to open them both at the same time. The flexion was executed, but alternately on the two sides; and it was the same in regard to the extension. She was not able to contract homologous muscles at the same time, whatever effort she made to obtain such a result. One could see that during the contractions she alternately fixed her attention upon the hand about to be called into movement. It was no longer possible for her to flex or extend simultaneously both forearms." This patient remained under observation for many months without any notable change in her condition.

The account given by Duchenne of this remarkable case was subsequently entirely confirmed by Briquet. He also holds that this extreme condition of anæsthesia of the muscles

is extremely rare. He, however, gives the details of another case,[1] just as extraordinary as that recorded by Duchenne, this patient having been under his care for nearly twelve months in 'la Charité.' Here are a few quotations extracted from his report:—

The patient was a girl, 21 years of age, suffering among other things from complete loss of taste, from noises in the ears, and from amblyopia especially on the left side.

"The skin of the whole periphery of the body is completely insensible; the patient absolutely does not feel anything which she touches.

"The muscles of the limbs do not feel pressure; they require to be pressed strongly against the bones before the patient feels anything. Passive movements are not at all felt, so that the patient has no consciousness of them if sight does not intervene.

"In short, the anæsthesia of the surface of the body is so extreme that, having bandaged her eyes, the patient has been taken from her bed with only her night-dress on, placed upon the floor of the ward where she has been left a few moments, and then replaced in her bed, without her having any consciousness of what had been done. The patient describes her condition by saying that she is like a balloon suspended in the air. She has only the sense of sight, which is weak, and that of hearing, which is just as bad.

"The muscles of the upper extremities are notably weak, nevertheless the patient can sew pretty well; she moves her fingers and hands well, but *if one prevents the intervention of sight no movement is any longer possible: whatever may be the efforts of the will, there is complete immobility.*

"The lower extremities are still more feeble; the patient can, whilst in bed, execute some movements of the whole limb, but she cannot move the toes, and when she is placed on her legs they immediately double under her, and there would inevitably be a fall [if she were not supported]."

After she had been in the hospital about four months this patient was in some respects better, she could for instance maintain herself in the standing position when supported on both sides, but as regards her knowledge of passive movements, and of the position of the limbs, there was no improvement whatever.

Fortunately I have been able to find the record of another

[1] 'Traité de l'Hystérie,' Paris, 1859, p. 304.

case recorded by Bazire,[1] very similar to those reported by Duchenne and Briquet. Only here we have to do with hemianæsthesia rather than with general insensibility, and there is the important fact that it occurred in a man rather than in a hysterical girl, and probably as a result of some slight organic lesion. The actual inability to move when the eyes were closed was here, moreover, limited to the lower extremity; though the power of moving the upper extremity under the same conditions was much more than usually affected. This case is in marked contrast with the ordinary run of cases of hemianæsthesia, in which there is generally no very appreciable loss of "muscular sense."

"W. P.——, aged 43, married, a plumber and gas fitter, was admitted into the Westminster Hospital, under Dr. Fincham's care, on April 25, 1865. He is tall, fairly nourished, with a sallow complexion, and there is a faint blue line along the edge of his gums. He has never had gout, rheumatic fever, or syphilis. A year ago he had an attack of lead colic, followed by dropping of the left wrist, which only lasted a few days.

"*Present state:*—There is complete *analgesia* of the whole left half of the body, exactly limited to the median line—namely, of the left half of the head, face, tongue, palate, neck, trunk, and penis, and the left arm and leg. Pricking and pinching, except in the spots to be presently mentioned, are obscurely felt as a mere contact, and many seconds after the impression is made. The left eyeball is so insensible to pain that the patient rubs it with impunity. The left half of the tongue cannot distinguish sapid substances, and the patient complains of a sensation of heat and dryness, and occasionally of pins and needles in that side of the organ. Instead of analgesia, there is tenderness on pressure in front of the left elbow-joint, and again near the lower edge of the deltoid. Differences of temperature are more acutely perceived on the affected rather than on the healthy side. There is nearly complete *anæsthesia* of the left half of the body, for when any point is touched on that side, the patient becomes conscious of it, only after an interval of several seconds, and besides, localizes the impression erroneously. He states that he feels as if through a thick layer of flannel or wool. He does not know the position of his left arm or leg, if he does not see it. *If, when his eyes are shut, he be asked to touch the tip of his nose with his left hand, he visibly makes considerable efforts, but his hand, which is raised with great difficulty and slowness,*

[1] Translation of Trousseau's 'Lectures,' 1866, p. 213.

either stops at a certain height, or goes to one side of the head, never succeeding in touching the nose. The movement is slow and hesitating, never abrupt or jerked. *When his eyes are shut, he cannot make the least movement with his left leg.* When he walks, he keeps his eyes fixed on the ground immediately before him, not on his legs. His gait is somewhat uncertain and unsteady, but not markedly so. *He cannot walk with his eyes closed;* he cannot stand, even with his eyes open, when his feet are closely approximated, and he looks straight before him. *If, while his feet are wide apart, he shuts his eyes, he immediately oscillates from before backwards, and threatens to fall down.* He says that he then feels as if he had only one leg.

"There is no real *diminution of motor power* at present, at least beyond a slight depression of the left angle of the mouth, and a scarcely perceptible deviation of the apex of the tongue to the left side. There is no difference of size between the muscles of the right and left limbs; electro-muscular contractility is perfect on both sides, but electro-muscular sensibility is almost *nil* on the left. None of the senses are affected, except *taste* in the left half of the tongue. Vision is good; the left pupil is, however, appreciably smaller than the right. Hearing is perfect on both sides. The intellect was never affected; memory is very good, and the man gives an excellent account of himself. There has never been any headache, but more or less giddiness throughout. Articulation thick and embarrassed, probably owing to the numbness of the left half of the tongue and soft palate; but the faculty of language is unimpaired. Appetite good; digestion easy; bowels at present regular, but were formerly very costive. The bladder was never affected.

"*Mode of Attack.*—The patient's illness dates from the beginning of September, 1864. It set in suddenly, without any premonitory symptoms, about seven o'clock one morning, with a sense of chilliness and numbness all down the left half of the body. He was not, however, prevented from attending to his usual occupation, until three weeks afterwards, when the numbness was replaced by complete insensibility. There was also some motor paralysis at first, as it appears from his statement; for he affirms that there was real and considerable weakness of his left arm and leg, that his left cheek was pendulous, and that the right angle of his mouth was pulled upwards and outwards whenever he spoke or laughed. Until he was examined by Dr. Fincham, he had not discovered his inability to move his left arm and leg unless he looked at them. Under the influence of faradization, and the administration of iodide of potassium for a short time, and afterwards of hypophosphite of soda, he gradually improved."

So far the cases to which I have referred have belonged to one or other of two categories, that is they have been either (*a*) cases of locomotor ataxy, or (*b*) they have been cases in which there has been loss of sensibility, deep as well as superficial, either unilateral or general. It is, indeed, now well known that these are the two forms of disease in which we are entitled to look very frequently for some loss or defect in kinæsthetic impressions generally, and of those pertaining to the "muscular sense" in particular. It is, however, also well known that all kinds of kinæsthetic impressions are by no means equally or necessarily affected in these two diseases. In each of them, for instance, ordinary cutaneous sensibility, and possibly even deep sensibility, may be gravely impaired or lost, when there is comparatively little loss of special "muscular sense" impressions.

Thus in locomotor ataxy it is now well-known that there is no regular parallelism between the loss of ordinary modes of sensibility, and the loss of special "muscular sense" impressions. In some rare cases of this disease there may be very little loss of ordinary modes of sensibility, and yet a marked deprivation of "muscular sense" impressions. The first case recorded in Landry's memoir seems to have been, in its early stages a well-marked one of this kind, and Trousseau said that out of about fifty cases of locomotor ataxy he had seen three of this latter type. This is a class of cases, however, about which we want further precise information.

Similarly, in regard to hemianæsthesia, it is now well known to be quite exceptional to meet with such cases as those recorded by Demeaux (p. 11), in which there was complete loss of special "muscular sense" impressions. The rule has been, for instance, with the hemianæsthetic patients which have been so thoroughly investigated by Charcot at the Salpêtrière that although there has been complete loss of tactile sensibility, and usually absolute insensibility to pain in the skin and all other sensitive structures on the affected side, together with slight paresis of the affected limbs, the so-called "muscular sense" has been nearly always preserved. Their ability to perform even complex muscular movements when the eyes are closed has remained quite intact. This

was well seen, for instance, in the case of one of his hemianæsthetic patients who was hypnotised by Charcot in the presence of a party of distinguished observers when the following phenomena were witnessed.[1]

"At 10.7, the patient being asleep, Professor Charcot told her to rise and take a chair. She did so, her eyes being closed and her eyelids tremulous. Having seated herself he told her to write her name. Pen, ink, and paper being furnished her, she sleepily wrote her own name, and afterwards, when ordered, Professor Charcot's, *her eyes remaining closed the whole time.* Whilst writing the skin over the right wrist was transfixed by a thick needle, but the patient appeared quite unconscious of the operation, continuing to write with the needle *in situ*. It is to be remarked that during the mesmeric state, the patient is anæsthetic on both sides of the body; whilst, in the waking state, it is only the left side which is anæsthetic. At 10.10 Professor Charcot told the patient to begin to sew. A half-hemmed towel was handed to her, and she at once commenced to sew with great dexterity and rapidity, co-ordinating admirably the movements of the two hands. The patient was then told to rise and go into an adjoining room used for photography; she did so, and on her way, having to descend a step, she walked with caution and safety, *her eyes being still closed.*"

Phenomena of the same kind, as well as others, were shown to Prof. Rouget of Montpellier and myself on the following day by Prof. Charcot, demonstrating that even in well-marked cases of superficial and deep anæsthesia, there may be no loss of the muscular sense or difficulty in executing even complicated voluntary movements when the eyes are closed. In a small minority of cases of hemianæsthesia, however, it is not so, and then we have to do with such phenomena as were met with in the typical case recorded by Demeaux if not such extreme results as those met with in the cases of Duchenne, Briquet and Bazire. We stand much in need of further records concerning this small minority of cases.[2]

[1] As recorded by Dr. Arthur Gamgee in 'Brit. Med. Journ.,' Oct. 12, 1878, p. 545.

[2] Such facts have been strangely lost sight of by Prof. Ferrier when seeking to interpret the results of his experiments upon the cerebral cortex of monkeys. It is difficult to understand, for instance, how he can make such statements as these ('Functions of the Brain,' 2nd ed., p. 64).—"There are certain clinical

Other cases, however, in which there is a marked defect in "muscular sense" impressions, appear to exist which cannot be included in either of the previous clinical categories. We must regard them as constituting a class apart, (*c*) the nature and clinical character of which have hitherto been extremely ill-defined. Landry recorded, but unfortunately in abstract only, two such cases. Brief though the details are, it will be well, however, to reproduce them here, as they are cases in which tactile sensibility was either unimpaired or but slightly affected.[1] The nature of the first case, more especially, is very obscure, owing to insufficiency of clinical details.

"Obs. IV.—The woman Reine, 42 years old, a seamstress, of a nervous lymphatic temperament, but strongly built, entered on March 20, 1852, the Beaujon Hospital (under the care of M. Sandras). After having experienced, in the course of the year 1849, headaches accompanied by giddiness, subjective noises, dizziness, and loss of appetite, she began to experience tinglings with a sensation of numbness in the upper and lower extremities;

---

facts which would seem to show that the sensibility of the muscles, and the muscular sense may continue though cutaneous sensibility is abolished. But, though the possibility of this cannot be denied, the actual existence of such a condition is far from being satisfactorily proved. Much of the evidence adduced is extremely unsatisfactory, and there is reason to believe that tactile sensibility is not entirely abolished, though there may be insensibility to pain and temperature, in the cases where the muscular sense really continues." Further on, after quoting some details concerning the rare and exceptional case of hemianæsthesia recorded by Demeaux, he says (p. 325), "These facts show clearly that the same condition which abolishes cutaneous sensibtlity, also entirely annihilates the so-called muscular sense," adding, "from such and similar cases, many of which I have myself seen and investigated, and multitudes of which are to be found in medical literature," etc. I should be extremely glad if Prof. Ferrier would give us some references to other cases like those of Demeaux, "multitudes" of which, as he says above, are to be found in medical literature. What he writes also on p. 344 (*loc. cit.*), seems to show that he considers it to be the rule that "muscular sense" is lost in cases of cerebral hemianæsthesia.

[1] These two cases, as well as those which follow (recorded by Zenner and Horsley) ought to prove interesting to Dr. Ferrier, considering the very positive views which he has recently expressed. Thus, he says ('Functions of the Brain,' 2nd ed., 1886, p. 380):—"There is no necessary connection between the power of directing movements and the muscular sense, as has been erroneously assumed by Brown Séquard and others. Loss of the muscular sense never occurs without general anæsthesia of the limb. No one has even furnished the slightest evidence of impairment, or loss of the muscular sense apart from profound impairment or total abolition of the common sensibility of the limb." Each of these statements is, in my opinion, thoroughly inaccurate.

at the same time her sight failed a little. In 1850 the lower extremities appeared to lose power, and her gait became staggering. From that time a paralytic affection developed which made continual progress.

"On her entry into the hospital (March 20, 1852), walking could not be accomplished without the aid of a stick. There was great difficulty in rising and in preserving her equilibrium; irregularity in the movements of the lower limbs; a tendency to assume an accelerating gait; power of executing all movements, which were extensive and energetic: *unconsciousness of passive movement; imperfect appreciation of the position of the lower extremities and of the extent of their active movements; defective measuring of movements when the eye does not watch them*, etc. A slight feebleness of the hands. *Sensibility absolutely intact.* Micturition and defecation normal. Integrity of intelligence and of all the senses excepting sight which is much enfeebled. No local morbid sign on the side of the nervous centres. . . . In February, 1853, there was an aggravation of all the symptoms. No relief had been derived from various kinds of treatment."

There is also no sufficient clue as to the nature of the next case. It occurred in a hysterical patient; though it seems very possible that it may have been due to a tumour in the right Rolandic area.

"Obs. V.—Angélique M. . . ., 46 years of age, a worker in furs, strongly built, but actually in a profoundly cachectic condition, of a nervous lymphatic temperament, entered, on Dec. 9, 1851, the Beaujon Hospital under the care of M. Sandras.

"This patient, subject to hysterical attacks for nearly twenty years, perceived at the end of 1849 that the left hand was becoming weak and numb. A paralysis of the whole limb soon developed, characterized by these symptoms: execution easy, though feeble, of all movements of the fingers, of the hand, and of the forearm, but without precision or being properly measured; *defective appreciation of passive movements and of the extent as well as of the energy of active movements; impossibility of appreciating weight.*

"All the functions of the upper limb are profoundly altered, and the patient cannot put it to any use which demands a little delicacy in the movement.

"*Sensations of contact and of temperature preserved*; sensations to pain obtuse and abolished in places. Sight very feeble; all the other senses are intact. No morbid local sign on the side of the nervous centres. Chloro-anæmic cachexia. Hysterical phenomena."

Another of these cases has, however, been recently recorded by P. Zenner[1] in which there is very strong reason for believing that the symptoms were produced by injury to Ferrier's so-called "motor centres" for the arm. The evidence in favour of this localisation is altogether similar in nature to that upon which Ferrier has himself relied, in several cases, in his lectures on the "Localisation of Cerebral Disease."

"A. N., age 23, shoemaker, was wounded during the court-house riot, March 29, 1884. The wound was on the right side of the head and laid bare the bone, on the surface of which a linear fracture, without any depression, was seen. It was made by a ball which had ploughed through the scalp and just grazed the bone. The wound was four inches long, and about two and a half inches from the median line, and would have been equally divided by a line passing from one auditory meatus over the vertex to the other.

"The man was unconscious for some hours after the injury, and there was occasional delirium for a period of two weeks. When consciousness returned it was observed that the left arm was entirely powerless, and that the face was drawn to the right side. No observation of the condition of the lower extremities was made. Shortly after the injury there was loss of sensibility in the hand; a pinch was not felt. From the second or third day, for about a week, there were frequent convulsive movements of short duration, apparently limited to the muscles of the face and unattended by loss of consciousness.

"Within a week power of movement at shoulder and elbow had returned, though the hand and wrist were yet powerless. After a few weeks a paresis of the left hand alone remained.

"I first saw the patient seven months after the date of the injury. The foregoing statements were obtained partly from the family, partly from Dr. N. P. Dandridge, who saw the patient about a week after the wound had been received. I saw the patient three times the latter part of October, and December the 1st, 1884, and May 16, 1885. As my first examination was a hasty one and no notes were taken, I will give a description from notes of my examination on December 1st.

"He is a man of average size, well built, and of good muscular development. On the right side of the head there is a linear cicatrix two and a half inches in length. The direction of the cicatrix is parallel with the sagittal suture, from which it is

[1] 'Journal of Nervous and Mental Disease,' New York, 1886, p. 438.

distant about two and three quarter inches. The central part of the cicatrix, in an antero-posterior direction, is just above the meatus auditorius. Patient does not suffer with headache, or other subjective symptoms, and, in his own opinion and that of his family, his mental powers are unimpaired. There is no impairment of sensation or motion anywhere except in the left upper extremity. He can walk long distances without difficulty, and the face, both in repose and action, shows no motor defect."

"*Left upper extremity.*—Power in the movements at the shoulder, and extension of the elbow about equals that of the right side. Cannot flex the elbow quite as forcibly as the right, but, perhaps, the difference is not greater than could be accounted for by its being the left arm, and its having been used less than the other for some time. Grasp in left hand fair, but quite appreciably less than in the right. But the difference in the power of the flexors of the fingers is more apparent when he attempts to flex one or several fingers separately. Then there is a decided difference in the two hands, the greatest difference being observed in the middle and ring fingers. This difference is also manifested when he holds an object, as a pencil, between his finger-tips. He holds it well with the left hand, but with decidedly less force than with the right.

"Cutaneous sensibility is good everywhere, excepting over the fingers. Elsewhere he feels the lightest touch (though in the left palm it is less accurately localized than in the right), but in the last two phalanges, both in their dorsal and palmar surfaces, he only becomes aware that the parts are touched when decided pressure is made. A small piece of coin, whose exact character is immediately recognized by the right hand, is often not felt at all when placed between the fingers of the left hand.

"*The knowledge of the position of the fingers is impaired.*—This is most marked in the middle and ring fingers. Occasionally he does not seem to know (eyes being closed) that the finger is moved. At other times he mistakes which finger it is. On the right side his knowledge in this respect is perfect.

"Actions requiring some delicacy of movement are performed very awkwardly. He buttons his coat with the left hand with the greatest difficulty.

"The only deep reflex action which could be elicited was a slight movement in tapping the lower end of the radius. The same reflex could not be elicited in the other side.

"A word more as to the improvement of the patient. According to the statement of the family, there was continuous improvement for six months, after which his condition appeared to remain

stationary. I first saw him seven months after he sustained his injury. As before stated, my examination at that time was hurried, and no notes were taken; but my impression is that both the impairment of sensation and muscular weakness were greater than they were at the time of my second examination, some five weeks later. But his condition May 16th (nearly fourteen months after the injury) was in no wise different from what it was December 1, 1884, unless it be that delicate movements, as buttoning his coat, were performed with more ease."

In this case there is strong reason for believing, as Zenner points out, that we have to do with a limited lesion of the cortex in the Rolandic area. The loss of "muscular sense" as well as the defect of tactile sensibility from a lesion so situated would, of course, be a matter of extreme importance. But the evidence in favour of such impairment of the "muscular sense" in particular, as a result of cortical lesions in the Rolandic area, is greatly strengthened by the following cases, extremely important in more ways than one, which have been recorded by Victor Horsley in a communication made by him to the recent meeting, in August last, of the British Medical Association.[1]

Case I.—James B., aged 22, was admitted into the National Hospital for Paralysis and Epilepsy, under the care of Dr. H. Jackson and Dr. Ferrier.

Past History.[2]—At the age of 7, the patient was run over by a cab, in Edinburgh. He was at once admitted into the Royal Infirmary, under Professor Annandale, who found a depressed comminuted fracture, with loss of brain-substance, in the situation mentioned below. The fragments of bone, etc., were removed, and the wound ultimately healed, although it suppurated freely, and hernia cerebri occurred. The patient was hemiplegic for some time, but gradually (seven weeks) the paralysis disappeared. At about 15 years, the patient began having fits, which were very intermittent. He was admitted into the hospital in 1885, when he had an enormous number of fits, and for some days was in the *status epilepticus*. After discharge from the hospital, he had

[1] *See* 'Brit. Med. Journal,' Oct. 9, 1886.

[2] Owing to the great courtesy and kindness of Prof. Annandale, who supplied Dr. Ferrier with an elaborate description of the case while under his care, I am fortunately able to furnish an accurate history of the injury causing the epilepsy. (V. H.)

no fit for seven weeks, at the end of which period they returned, and for three days before admission he was again in the *status epilepticus.*

Present state.[1]—On the left side of the vertex of the head (the exact site, as determined by measurement, being the centre of the upper third of the ascending frontal convolution; that is, posterior to the hinder end of the superior frontal sulcus) there was a quadradiate scar, opposite the centre of which the bone could be felt to be wanting, so as to form an oval opening in the skull, the long diameter of which was about one inch, and parallel to the sagittal suture. Pressure on this scar always gave pain, which was very greatly increased when the patient was suffering one of his paroxysms of fits.

Fits.—The fits, which occurred in batches (at this time the patient had 3,000 in a fortnight), were almost always of the same character, usually commencing in the right lower limb, sometimes in both the right limbs simultaneously. An example of a fit of the first category is as follows:—

"The right lower limb was tonically extended, and the seat of clonic spasm. The right upper limb was then slowly extended at right angles, to the body, the wrist and fingers being flexed; the fingers next became extended, and clonic spasms of flexion and extension affected the whole limb, the elbow being gradually flexed. By this time, spasms in the lower limb having ceased, but those in the upper limb continuing vigorously, spasm gradually affected the right angle of the mouth, spreading over the right side of the face, and followed by turning of the head and eyes to the right."

To sum up, the parts affected were so in the order of lower limb, upper limb, face, and neck; the character of the movements was, first, extension, then confusion, finally, flexion,[2] showing clearly that the focus of discharge was situated around the posterior end of the superior frontal sulcus, this point coinciding, as mentioned above, with that found by actual measurement. Before going on to describe the surgical treatment, it is important to mention that the patient was distinctly hemiplegic, even ten days after the last fit, but he could perform all the movements of the right limbs, though about half as strongly as on the left side, there was no affection of sensation on the right side, while the reflexes, superficial and deep, were exaggerated in both the right limbs.

[1] Only those facts are given which directly bear on the cerebral disturbance.

[2] See a paper by Dr. Beevor and myself, to be shortly published by the Royal Society in the 'Philosophical Transactions.' (V. H.)

Operation, May 25th, 1886.—According to the method described in the foregoing paper, the bone around the old opening was freely removed, the dura mater, arachnoid, and skin being found to form a homogeneous mass of fibrous tissue; the former was raised with the flap. The scar in the brain was found to be highly vascular, of a deep red colour, and about three centimètres long and two broad. The membrane covering the brain around appeared to be very opaque, and the brain of a slightly yellower tinge than usual. The scar, and about half a centimètre of surrounding brain-substance, was excised to the depth of two centimètres. It was then found that the scar-tissue penetrated a few millimètres further into the corona radiata fibres of the marginal convolution. This portion was then removed, and the wound closed. In the removal of the mass, three fair-sized veins, coming directly from the middle of the area for the upper limb, had to be ligatured, since they passed directly into the scar. The wound completely healed in a week. The tension of serum was twice relieved (once, probably unnecessarily). The most interesting point now to be recorded is, that after the operation the patient was at first completely paralysed in digits of the right upper limb; and for further flexion of the wrist and supination of the fore-arm. Coupled with this motor paralysis, there was loss of tactile sensibility over the dorsum of the two distant phalanges of the fingers. He could not localise the touch anywhere below the wrist within the distance of one internode; finally, he could not tell the position of any of the joints of the digits. Then we have here, apparently, a distinct instance of loss of tactile sensibility and muscular sense, coupled with motor paralysis, all due to lesion of the cortex[1]. It cannot, however, be too clearly understood that it is very possible that some of the fibres coming from the gyrus fornicatus in the corona radiata may very probably have been injured. This condition of motor and sensory paralysis gradually disappeared in the course of the next two months. Up to the present time the patient has had no fits.

Case II.—Thomas W., aged 20, was admitted into the National Hospital under the care of Dr. Hughlings Jackson.

Family History.—There was nothing important, save that a paternal aunt died of consumption.

Past History.—He had had many attacks of pleurisy after the age of 15.

History of Present Illness.—He began, in January, 1884, to have "cramps" in the left thumb and forefinger, these consisting of

[1] By this I mean, of course, the disturbance in the area for the upper limb produced by the ligature of the veins coming from it, as noted above. (V. H.)

clonic opposition of the named digits, and occurring about twice a day for three months. The first severe fit occurred in March, 1884. Spasm spread up the arm, and the patient fell. He had the second in January, 1885. Then followed a series of remissions of the twitchings, until, in August, 1885, another severe fit commenced a series of fits, occurring once or twice a week, until admission on December 4th, 1885. The character of the fits was almost always the same. They began by clonic spasmodic opposition of the thumb and forefinger (left), the wrist next, and then the elbow and shoulder were flexed clonically, then the face twitched, and the patient lost consciousness. The hand and eyes then turned to the left, and the left lower limb was drawn up. The right lower limb was next attacked, and, finally, the right upper limbs. Paralysis of the left upper limb frequently followed a fit. At frequent intervals every day the patient's thumb would commence twitching, but progress of the convulsion could often be arrested by stretching the thumb or applying a ligature. In February and March, 1886, the twitchings frequently commenced in the face, but in April again the thumb was the most frequent seat of origin of the fits.

Present State (much abbreviated). *Motion.*—The grasp of the left hand was 45; of the right, 85. He could perform all movements with the left upper limb, though those of the hand were rather enfeebled. The left thumb was frequently in a state of rigidity, alternating with clonic spasm. (This state could easily be induced by manipulating the thumb.) *Sensation.*—There was no affection, save loss of muscular sense (that is, sense of position, etc.) in the left thumb. *Deep Reflexes* were exaggerated in the left upper limb. The patient frequently had severe headache, beginning at the occiput, and shooting forward, especially to the right parietal region. The *Optic Discs*, examined by Mr. Marcus Gunn, appeared to be normal, though very pink (physiological hyperæmia).

Diagnosis.—In a paper referred to in the foregoing case (No. I.), Dr. Beevor and myself have shown, that the movement of opposition of the thumb and finger can be elicited by minimal stimulation of the ascending frontal and parietal convolutions at the line of junction of their lower and middle thirds. Dr. Hughlings Jackson witnessed one of our experiments demonstrating this fact, and expressed his belief that this patient (Case II.) was suffering from an irritative lesion of unknown nature, situated in the part of the brain thus indicated. Confirmation of this opinion was to hand in the order of march of the spasm, which was in nature and arrangement in exact accord with

the results of the investigations referred to. An exploratory operation was therefore decided upon.

Operation (June 22nd, 1886).—The seat of the lesion having been determined by measurement, the large trephine was applied; and, on raising the dura mater, a tumour came into view. More bone was removed above and in front, so as to completely expose the mass to which the dura mater was adherent. The border of the tumour stood out about one-eighth of an inch from the surface of the brain, and it was much denser than the brain-substance. It appeared to be only half an inch broad, but as the brain-substance all round it for more than half an inch appeared dusky and rather livid, I removed freely all the part apparently diseased. (As is shown in the photograph and specimen, this procedure was fully justified, since the growth spread very widely under the cortex.) Before closing the wound, the centre of the thumb-area was removed by free incision. This detail Dr. Jackson and myself had resolved to carry out in the possible event of there being no obvious gross organic disease, in order to prevent, as far as possible, occurrence of the epilepsy.[1] Numerous vessels were ligatured, especially three or four at the upper border of the growth proceeding from the rest of the cortex, for the movements of the upper limb. The wound was closed as before. Five-sixths of it healed by the first intention in a week, in spite of the fact that there was considerable œdema of the scalp, due to irritation by the carbolic gauze (removed speedily by changing to eucalyptus gauze). The remaining sixth, just at the lower border of the flap, gave way, and healed by granulation, after separation of a small piece of skin at the edge. The after-condition of the patient was most interesting and important. There was, next day, partial motor paralysis of the left side of the face (lower division), complete motor paralysis of the left upper limb, from and including the shoulder. On the 27th June, there was noted left hemianæsthesia to a light touch (sensibility to pain unaltered), localisation of a prick of a pin very deficient all over the left side, perfect on the right, complete loss of muscular sense in the left upper limb below the shoulder. On the second day after the operation, when making an effort to move the left upper limb, the patient suddenly put his hand to the wound, and said he felt a

[1] To discuss this point in detail would entail excessive lengthening of this paper; but I wish to point out that, as strongly urged by Dr. Jackson, the removal of an epileptogenous focus is not only justifiable, but called for. The exact localisation could be ascertained by the use of the induction current, the employment of which means I resorted to for diagnosis nearly three years ago. 'Brain,' Vol. VII., p. 232. (V. H.)

"buzzing" in the head there. When the left upper limb was passively moved, he also complained that pain seemed to shoot up the "bones of the limb," side of the neck, and through the hand to the wound. The deep reflexes were much exaggerated on the left side in both limbs. All the above conditions gradually improved, and at the date of the meeting, the patient had regained everything, except that the grasp of the left hand was not quite so good as before, and the five movements of the fingers remained hampered. Further, the deep reflexes on the left side are at the present time still very much exaggerated. The patient had, in July, a few slight twitches in the three right fingers; none in the thumb or index; no fits since operation. The tumour was composed of dense fibrous tissue, with two caseated foci, microscopical examination proving it to be tubercular.

It is unfortunate that in the first of these cases no observations were made upon the state of the lower extremity in regard to its "muscular sense" impressions. Such cases require to be examined with the greatest care in order to obtain trustworthy information concerning the "muscular sense." We must look therefore for further information in these directions, and no cases will be so important as those in which portions of the Rolandic area have been actually excised by the surgeon. They are veritable experiments, from one point of view, calculated to throw light in directions from which none is to be hoped for from experiments upon animals not endowed with the gift of speech, in regard to the presence or absence of "muscular sense" impressions, and, it should be added, even of tactile impressions in the hand in cases, for instance, where corresponding portions of the Rolandic area have been removed by operation.

A very important paper by Westphal in reference to some of these questions, is to be found in the "Charité-Annalen"[1] concerning a case of wasting disease in the ascending parietal convolution and parietal lobule (though not absolutely limited thereto) in which, in addition to other troubles, there was the association of paralysis with marked loss of "muscular sense," and some diminution in tactile sensibility.

It seems best to defer, for the present, any further remarks

[1] VII. Jahrg., 1882.

as to the paths or cortical termini for "muscular sense" impressions.

Meanwhile, what has already been brought forward will show that three kinds of defects related to one another, it is true, are to be looked for in cases where special "muscular sense" impressions are either absent or defective. These defects are as follows:—

(1) Defective knowledge of the extent of movements executed, either actively or passively, as well as of the position of the limbs, when the eyes are closed.

(2) Difficulty in discriminating differences in weight or degrees of resistance when muscles are called into play.

(3) Difficulty in accurately performing given movements when the eyes are closed.

It is, of course, of some importance to know whether these three kinds of disability are referable to loss of kinæsthetic impressions generally or whether they are specially related to loss of the most important group of these kinæsthetic impressions, viz., those of the "muscular sense."

This subject has already been alluded to in part; it has been stated for instance, that in the majority of hemianæsthetic cases, there is (3) no difficulty in accurately performing movements when the eyes are closed. This is a matter of common observation, but it has been thoroughly established of late years by Charcot and others.

Again, it has been shown more especially by the observations of Leyden [1] and Bernhardt,[2] that the sensibility of the skin may be partially or completely destroyed without appreciably altering (2) the patient's ability to discriminate differences in weights when his muscles are called into play. It may be stated that in applying this test to the upper extremities, leather balls of the same size but differently weighted within with lead may be used, or where these are not to be had we may roughly test the ability to discriminate differences in weight (the patient's eyes being closed) by placing piles of pence, or books of the same size except as regards thickness, upon the extended palm. In the examination

[1] 'Archiv de Virchow,' t. 47, p. 325, *et seq.*
[2] 'Archiv für Psychiatrie,' iii., pp. 618 and 632.

of the lower extremities different weights may be suspended from the extended limbs. On this subject Bazire[1] has said:—

"Quite recently Dr. Jaccoud has applied Weber's method to the lower extremities, and the results of his experiments, made on twenty-four different individuals, tend to show that it is possible, in health, to distinguish on an average a difference of from 50 to 70 grammes (say about two ounces) between two weights successively suspended from a lower limb. In six ataxic patients to whom this test was applied, the minimum of the difference in weight appreciated by the patient was found by Dr. Jaccoud to be considerably above the healthy average, varying from 100 to 3000 grammes (from 3¼ ounces to four pounds)."

Spaeth[2] also, without being aware of the experiments just cited, adopted a similar method to investigate the state of the muscular sense in two cases of ataxy, and in both he found a notable diminution in the individual's discriminative power—indeed, in one of the two cases no difference was perceived unless the successive weights bore the ratio to one another of 1 to 100.

There can be no doubt that some of a patient's ability (3) to discriminate the extent of active or passive movements executed, as well as some of his knowledge of the position of his limbs, depends upon kinæsthetic impressions coming from the skin and from joints. Some have supposed that all this kind of knowledge comes from these ordinary kinæsthetic impressions.

Wundt, for instance, thinks that these sensations belong to a different category from those by which we appreciate weight or resistance.[3] Gley[4] also seems to think that the knowledge of the position and movements of the limb is derived in the main, if not wholly, from skin and joint impressions.

This, however, is in my opinion an erroneous conclusion, based upon the examination of one of the exceptional cases of hemianæsthesia, like that recorded by Demeaux (p. 11). A study of the two cases last quoted from Landry[5] shows conclu-

[1] Translation of Trousseau's 'Lectures,' 1866, p. 212.

[2] Beitr. zur Lehre von den Tabes Dorsalis. Tübingen, 1864.

[3] *Loc. cit.*, t. i., p. 426.

[4] Rev. philosoph., December, 1885, p. 605.

[5] Obs. iv. and v. (see pp. 23 and 24.)

sively that knowledge as to the position and movements of the limbs may be more or less lost even when cutaneous sensibility is not at all or but little impaired. Other good clinical evidence may also be adduced pointing in the same direction. Thus Bazire, in reporting a case of locomotor ataxy, says[1] :—"His gait was very characteristic of locomotor ataxy. . . Within the last few weeks he had noticed that he could not tell the position of his limbs in bed, under the bed-clothes. He still complained of numbness of the legs, and yet, strangely enough, when tactile sensibility was tested with the æsthesiometer, it was found unimpaired, for he could feel the two points of the instrument at a distance of only two inches, when applied in front of the legs." In cases of this kind, it could only be expected that there would be a defective knowledge of the position of the limbs while the limbs are at rest, in bed. As soon as they are moved, when sensibility is either intact or but little impaired, the accompanying superficial sensations must necessarily give a pretty accurate knowledge as to their position. This source of difficulty is present, therefore, in all cases in which superficial insensibility does not exist though loss of the muscular sense is suspected; we cannot well move the limb or parts of the limb (such as the fingers) about at random, with the view of asking in what exact position they are ultimately left, without giving the patient *some* aid through his intact sense of touch. Of course if, in such a case, we still find that the patient has a defective appreciation of the position in which his limbs are ultimately left, the evidence is all the stronger that his "muscular sense" must be defective.

It thus seems clear that of the three disabilities above named, the second and third are dependent almost if not entirely upon loss of the muscular sense, whilst the first (the knowledge of the extent and direction of passive movements, and of the position of limbs) is very greatly due also to "muscular sense" impressions, though it is in part undoubtedly derived from other less special kinæsthetic impressions.

In regard to the very extraordinary cases recorded by Duchenne, Briquet, and Bazire, in which the third of the above-named disabilities was carried to such an extent that

[1] Trousseau's 'Lectures,' translated by Bazire, part i., case vi., p. 195.

even the simplest movements could not be performed at all without the aid of the sense of sight, it seems to me that we have here to do with functional defects in the cortical termini for "muscular sense" impressions, as well as interference with the functional integrity of the afferent channels for such impressions. These latter are alone affected (in concert with the afferent channels for other sense impressions) in those more ordinary severe cases of hemianæsthesia of that type recorded by Demaux. But in these altogether extraordinary cases of Duchenne and Briquet there existed, I think, in addition a low functional activity of the "muscular sense" centres[1]; that is to say, these centres could not be roused into activity by comparatively weak associational stimuli (as during volition with eyes closed); though they were able to respond under the influence of a stronger stimulus from the visual centre. May we not have in certain *hysterical paralyses of cerebral type* a still greater functional degradation of these "muscular sense" centres? Such patients do not move the affected limb or limbs either when their eyes are open or closed.

We may find illustrative, though not exactly parallel, conditions among speech defects of an amnesic type, due to a lowered functional condition of the auditory centre. When these, for instance, cannot be roused into proper activity by ordinary associational stimuli, the patient, as a consequence, cannot recall the words that he wishes; and, as a further consequence, cannot utter such words. This is a kind of paralysis, so far as these speech movements are concerned. On the other hand, such a patient may be able to repeat at once words uttered before him by another person. The affected auditory centre in this case reacts to the stronger stimulus; just as I imagine the affected "muscular sense" centres reacted in the cases of Duchenne, Briquet, and Bazire, under strong associational stimuli coming from the visual centres.

Supposing, however, the left auditory centre to be more damaged still, either functionally or structurally, speech becomes impossible. Speech is paralysed, in fact, in such a case, if the degradation of the auditory centre be of functional type, in just the same way as I suggest a hysterical patient is

[1] See 'Brain as an Organ of Mind,' pp. 616 and 621.

paralysed when she is suffering from a very low functional condition of her "muscular sense" centres for leg or arm, or both.

It is extremely interesting to find, in the case of Bazire, the complete inability to move the limb without the stimulus of sight impressions, existing only in the leg, while in the arm there was an approximation to this condition.

A fuller understanding of my views, in regard to these very interesting cases, may be expected after the reader has studied a subsequent section of this paper on the part taken by "Muscular Sense Impressions in the Execution of Voluntary and Automatic Movements" (p. 52).

Having said thus much concerning kinæsthetic impressions as a whole, and "muscular sense" impressions in particular, it may now be well to endeavour to show that both kinæsthetic impressions as a whole and these "muscular sense" impressions in particular, are, like other sensations, capable of being revived in idea. It seems desirable to advance something like a formal proof of this here, as, in our opinion, these ideal re vivalsof kinæsthetic impressions aid in the performance of most important functions—that is, in the execution of all the voluntary movements we perform.

Just as the blind man can recall to mind previous sight impressions, or the deaf man sounds previously heard, so can the man who has had an arm amputated call to mind movements which this limb had formerly executed, by reviving in idea the kinæsthetic impressions previously associated therewith. Many remarkable instances of this have been recorded by Weir-Mitchell in his work entitled 'Injuries of Nerves and their Consequences' (p. 348, *et seq.*). I am inclined to think that in very many of these cases the conjuring up of subjective impressions of the several kinds mentioned is more facile than in persons who are neither blind, deaf, nor armless; and that, in many instances, the impressions so revived, assume such an intensity as to appear more like hallucinations than ordinary sensory revivals. Weir-Mitchell says:—"Nearly every man who loses a limb carries about with him a constant or inconstant phantom of the missing member, a sensory ghost of that much of himself. . . . The sensation of the presence of

the part removed exists in many persons as soon as they come from under the influence of the anæsthetic used at the time of the amputation, but in others it only arises after they cease to suffer pain, being rarely delayed beyond three weeks. The more healthy the stump, the less perfect after a time becomes the sense of the existence of the limb removed, while it is liable to be recalled by a blow or anything which causes a return of subjective sensation. . . . Even in those who are least conscious of the missing part, I have amazed them by suddenly recalling it with the aid of a faradic current applied to the nerves of the stump. It is not easy to forget the astonishment with which some of these persons re-awaken to a perception of the long lost leg or arm."

In regard to the ideal recall of movements of the missing member, Weir-Mitchell says:—"We find that in a very small number there is no consciousness of power to stir any part of the absent members by force of will. All others are able to will a movement and apparently to themselves to execute it more or less effectively, although in most of the amputated such phantom motions are confined to the fingers or toes, which rarely seem to possess the normal range either of flexion or extension. Yet the certainty with which these patients describe the limitations of motion, and their confidence as to the place assumed by the parts moved, are truly remarkable; while these restricted movements are pretty surely painful, and the effort is apt to excite twitching in the stump. . . . A small number have entire and painless freedom of motion as regards all parts of the hand. 'My hand is now open, or, it is shut,' they say. 'I touch the thumb with the little finger.' 'The hand is now in the writing position,' etc. Between these cases and such as are conscious of an immobile member, every grade of difference as to motion is to be found."

Another very remarkable fact of some importance is that, according to Weir-Mitchell, such sensations may be roused at will and even revived after they have been in abeyance for years, by stimulation of afferent nerves. He says:—"If we faradise the track of the nerve in or above the stump, we may cause the lost fingers and thumb to seem to be flexed and extended, and, what is most remarkable, parts of which the

man is conscious, but which he has not tried to stir for years, may thus be made to appear to move, to his utter amazement. In one case I thus acted on the nerves, so as to cause a thumb which for years was constantly and violently bent in on the palm to straighten out completely. On breaking the circuit without warning, the patient exclaimed that his thumb was cutting the palm again, and the same result was obtained by shifting the conductors so as to put the nerves out of the circuit. . . . In a case of amputation at the shoulder-joint, in which all consciousness of the limb had long since vanished, I suddenly faradised the brachial plexus, when the patient said at once, 'My hand is there again. It is bent all up and hurts me.' These impressions are correctly referred by the patient, so that faradisation of the musculo-spiral, or the ulnar, gives sensations of movement in the related parts."

These effects of faradisation of the nerve trunks are very interesting, and are entirely in accordance with the statements previously quoted to the effect, that the more healthy the stump the less perfect, after a time, becomes the sense of the existence of the limb that has been removed. If a mere blow upon the stump tends to recall such a sensation, how much more should a definite stimulus applied to the very nerves themselves. These facts simply mean, that the kinæsthetic centres in relation with a lost limb are apt to drop into a condition of functional inertia, but that they are easily roused from this state by the advent of afferent stimuli, and that they are all the more powerfully roused when the stimulus applied is in itself powerful. In these statements, however, we are a little taking for granted the correctness of a view as to the nature of these impressions which has not yet been formally considered (see p. 42), although some of the strongest evidence in its favour has already been adduced in preceding pages.

In regard to the varying power of recall of kinæsthetic impressions exhibited by different persons whose limbs have been amputated, this may be due, in part, to varying conditions of the stump as above indicated; and perhaps more especially to varying states of the nerve ends, and the extent to which they are invaded by processes of sclerosis. Beyond all such causes of difference, however, we may expect that

there would be naturally in different individuals, great variations in the power of recalling kinæsthetic impressions, just as an extreme variability is met with in different persons in regard to their power of recalling visual or auditory impressions, as F. Galton has so fully shown in the case of the former. A few instances of such variations will well illustrate this part of our subject.

Galton made a series of careful enquiries to ascertain to what extent different persons are endowed with the power of recalling, or seeing with the mind's eye, distinct images of objects in their natural grouping and colouring, taking as a trial-subject the person's own breakfast table as he or she sat down to it in the morning. The one hundred answers which he received disclosed an extraordinary range of variation in this respect among different individuals, as may be seen by the perusal of his interesting essay on 'Mental Imagery.'[1] One of those who had the highest power of recall answers:—"Thinking of the breakfast table this morning, all the objects in my mental picture are as bright as the actual scene." One of the worst answers:—"No individual objects, only a general idea of a very uncertain kind." While the person who possessed the lowest power of all in this direction, answered:—"My powers are zero. To my consciousness there is almost no association of memory with objective visual impressions. I recollect the breakfast table but do not see it." Other very interesting examples of variation in this power of recalling visual images are cited. Thus Mr. Galton says:—"One statesman has assured me that a certain hesitation in utterance which he has at times, is due to his being plagued by the image of his manuscript speech, with its original erasures and corrections. He cannot lay the ghost, and he puzzles in trying to decipher it. . . . A distinguished writer on metaphysical topics assures me that he is exceptionally quick at recognizing a face that he has seen before, but he cannot call up a mental image of any face with clearness. . . . There are a few persons in whom the visualising faculty is so low that they can mentally see neither numerals nor anything else; and again there are a few in whom it is so high as to give rise to hallucinations."

[1] 'Inquiries into Human Faculty,' 1883, p. 83.

With variations such as these to be met with in regard to the impressions of a vivid sense like that of sight, it is much more easy to explain, by the principle of individual variation, some of the remarkable differences in the power of recalling kinæsthetic impressions met with among the persons examined by Weir-Mitchell.

The cases to which we have just been referring are instances in which kinæsthetic impressions as a whole are revived. There is, however, good evidence to show that "muscular sense" impressions in particular are also capable of being revived in idea. It has been already shown that these are the impressions which almost, if not quite alone, are concerned with the estimation of weight or resistance. If such impressions, however, had not been capable of being revived in idea, we should have no power of discriminating differences in weights when they are lifted in succession, and still less should we have that power which we possess of lifting certain bodies and saying at once what is their approximate weight.

In the former case we have mentally to compare our present feelings of resistance with our memory of similar impressions experienced a few moments before; whereas, the fact of our possessing such powers as are referred to in the second case, shows, that we are capable of acquiring ingrained "standards of weight" by the constant exercise of this faculty, dependent of course upon our power of ideally recalling such impressions, so that at last we are enabled to judge intuitively as to the approximation of bodies to certain ideal standards of weight, just as we are enabled to judge of the approximation of certain presently perceived colours or sounds to certain "standard" visual or auditory impressions. In regard to this point Professor Bain says:[1]—"Absolute weight implies a permanent standard, and a permanent impression of that standard. When I lift a weight, and pronounce it to be seven pounds, I make a comparison between the present feeling and the impression acquired by handling the standard weight of seven pounds or things equivalent thereto. This absolute comparison, therefore, implies the enduring and recoverable sensibility to impressions of resistance, which is also a fact of the human

[1] 'The Senses and the Intellect.' 3rd ed., p. 93.

constitution. . . . A receiver of posted letters contracts an ingrained sensibility to half an ounce, and can say, of any letter put into his hand, whether it produces a sensibility equal to or under the standard."

But it is one of the fundamental positions of physiological psychology which Professor Bain has strongly enforced, that a renewed feeling or idea must depend upon the action of precisely the same parts as the original feeling. On this head he says[1]:—"What is the manner of occupation of the brain with a resuscitated feeling of resistance, a smell, or a sound? There is only one answer that seems admissible. The renewed feeling occupies the very same parts, and in the same manner as the original feeling, and in no other parts, nor in any other assignable manner. . . . For where should a past feeling be re-embodied if not in the same organs as the feeling when present? It is only in this way that its identity can be preserved; a feeling differently embodied would be a different feeling."

The Nature and Origin of "Muscular Sense" Impressions.

Almost all that has been written by others in regard to what I have in this paper spoken of as "kinæsthetic impressions," has been brought forward in relation with the several writers, views concerning the existence and nature of that power in us whereby we are enabled to judge of "resistance" or "weight." This power has been discussed by them under such different names as these:—"muscular sense" (Sir Charles Bell); a "sense of force" (E. H. Weber); a "sens de l'activité musculaire" (Landry); a "conscience musculaire" (Duchenne); a "locomotive faculty" (Sir William Hamilton); the "feelings of movement" (Bain); and the "sense of innervation" (Wundt).

Some of these writers, such as Sir William Hamilton[2] and Wundt,[3] have carefully discriminated the different components of the group of impressions accompanying and resulting from

[1] 'The Senses and the Intellect,' 3rd ed., p. 338.

[2] 'Notes and Dissertations on Reid.'

[3] 'Élements de psychol. physiol.,' 2nd ed. (trad. franç.), 1886, t. i., pp. 421–426.

movements; others, like Landry[1] and Bain[2] have not always been careful to make such discrimination, but have spoken rather as though all the impressions derived from movement might be included under that endowment which others speak of as the "muscular sense"—a laxity which is all the less likely to recur if the term kinæsthetic should be adopted as a general designation for the whole mixed group of impressions which accompany movement.[3]

From a psychological point of view the highest interest has attached to these discussions, seeing that the sensations of "resistance" which we derive through the exercise of our muscles have been supposed to give us a knowledge altogether apart from that derivable through so-called "passive sensations"—an immediate knowledge, in fact, of an external world or "non-ego."

Those who consider that through the performance of movements we acquire a kind of knowledge which is altogether peculiar, are not precisely agreed among themselves as to the exact mode in which they so acquire it. These, however, are altogether minor differences, and as a class the holders of such views must be separated from another group of psychologists and physiologists who maintain that there is nothing intrinsically peculiar in the sensations and impressions constituting that combination of feelings which comes to us as a consequence of movement.

Omitting minor detail, therefore, we have two points of view in reference to the nature and origin of the impressions whence we derive our knowledge of "resistance" and of

[1] 'Mém. sur la paral. du sentim. d. activ. musc.' 1855, p. 262.

[2] 'The Senses and the Intellect,' 3rd ed., p. 82.

[3] This looseness of phraseology is, in fact, still very common, as may be judged from the following quotation from Ferrier, where he is speaking of the views of Hitzig and Nothnagel. He says (*loc. cit.*, p. 379): "The term muscular sense, as ordinarily understood, and as employed by these authors, is applied to the assemblage of centripetal impressions generated by the act of muscular contraction in the muscles themselves, as well as in the skin, fasciæ, ligaments, and joints." Historical references bearing upon the subject of the "muscular sense" generally, are mainly to be found in the following three places, but especially in the last of them:—Hamilton's 'Notes and Dissert. on Reid'; Bastian's 'Brain as an Organ of Mind,' *Appendix*; and Sternberg, in Pflüger's 'Archiv f. Physiol.' Bd. xxxvii.

"weight," whereby we acquire our knowledge of the different degrees of "consistence" of bodies, and (through consciousness of the nature and extent of our movements) whereby we learn the differences in "form" which bodies present.

These conflicting views must now be briefly but definitely stated, in order that it may be made perfectly clear which of them is in accordance, and which at variance, with facts that are now well established:—

(1.) According to the first view our notions of "resistance" are not derived from sensations at all, but are due to a central psychic process (to which Wundt has applied the term "sense of innervation"). In reference to such a view Ludwig says:—"It is conceivable and not unlikely, that all knowledge and discrimination arrived at through the exertion of the voluntary muscles are attained directly through the act of voluntary excitation; so that the effort of the will is at once proceeded on as a means of judgment." On this subject also, J. Müller said:—"It is not certain that the idea of the force employed in muscular contraction depends solely upon a sensation. We have a very exact notion of the quantity of nerve-force starting from the brain which is necessary to produce a certain movement. . . . It would be very possible that the appreciation of the weight and pressure in cases where we raise or resist, should be in part at least not a sensation in the muscle, but a notion of the quantity of nerve-force which the brain is excited to call into action." According to Wundt, "the strength of the sensation is dependent only on the strength of the motive influence passing outwards from the centre, which sets on the innervation of the motor-nerves." Bain, again, holds "that the sensibility accompanying muscular movement coincides with the outgoing stream of motor-energy, and does not, as in the case of pure sensation, result from an influence passing inwards, by ingoing or sensory nerves," or, as he elsewhere says, "we are bound to presume that this is the concomitant of the outgoing current by which the muscles are stimulated to act." Hughlings-Jackson, amongst ourselves, has always been a strong upholder of these views.

(2.) The opposite view is not so old. The earliest statement that I have seen to the effect that the sensations accompanying

or resulting from movement are afferent and not efferent feelings, is that of Lotze, who says:[1]—"We must affirm universally that in the muscular feeling we are not sensible of the *force* on its way to produce an effect, but only of the *sufferance* already produced in our moveable organs, the muscles, after the force has, in a manner unobservable by us, exerted upon them its causality." This view was, however, very shortly after, much more clearly expressed by Landry[2] in these terms: —"The *ego* has a direct consciousness of the phenomena of volition; it knows immediately that there has been a voluntary stimulus, and to what part of the body it has been directed; as to the effects produced, it is only mediately informed of these, and can disregard them. . . . The nervous action which incites the movement can only, therefore, furnish to consciousness an idea of the volition, and not that of its execution; besides, if the sensorium has a knowledge of the excitation of the excito-motor faculties, it ignores the quantity of the nerve action exerted. It is necessary that the effect of the central incitation (the contraction) should be produced in order that the brain may perceive, and then it perceives at the same time both the seat and the degree of contraction. The movement is, therefore, the source whence we derive notions of this kind."

In 1869 I expressed the view,[3] that "*all* the information which Prof. Bain considers to have been revealed to us by the activity of our locomotive organs through an 'active' mode of sensibility, has been derived from modes of passive sensibility (occasioned, it is true, by this activity), and from inferences which have gradually been built up upon these . . . The feeling of 'expended energy,' therefore, by which we obtain our ideas of resistance and of an external world, is not contained, as we think, in the volitional act itself, but is derived from impressions emanating from the moving organs themselves during the actual accomplishment of movements." And after referring to the clinical evidence that was available

[1] 'Medicinische Psychologie,' 1852, p. 293.

[2] 'Traité des paralysies,' Paris, 1859. Similar views were indeed expressed in his memoir in the 'Gaz. des Hôpitaux,' 1855.

[3] "On the 'Muscular Sense,' and on the Physiology of Thinking." 'British Medical Journal,' April, 1869.

at that time, concerning cases in which the so-called "muscular sense" was supposed to be more or less defective, I arrived at the following conclusion:—"There is clearly a loss of something in these cases, of a something which serves as a guide in the execution of voluntary movements, but whose absence can be compensated by the supervision of the visual sense; and this is, in great part, the function which some physiologists attach to the 'muscular sense' . . . my position is that these impressions of the muscular sense, whose existence we are thus obliged to postulate, are *unconscious*[1] impressions, and that the conscious impressions that have usually been stated to fall within its province are really derivable through modes of ordinary cutaneous and deep sensibility."

In 1880, whilst adhering to almost precisely the same views, I maintained[2] that the time had come when it would be advantageous to speak, in lieu of a "muscular sense," of a "Sense of Movement, as a separate endowment, of a complex kind, whereby we are made acquainted with the position and movements of our limbs, whereby we judge of 'weight' and 'resistance,' and by means of which the brain also derives much unconscious guidance in the performance of movements generally, but especially in those of the automatic type. . . . There are included under it, as its several components, cutaneous impressions, impressions from muscles and other deep textures of the limbs (such as fasciæ, tendons, and articular surfaces), all of which yield conscious impressions of various degrees of definiteness; and in addition there seems to be a highly important set of 'unfelt' impressions, which guide the motor activity of the brain by automatically bringing it into relation with the different degrees of contraction of muscles that may be in a state of action."

Meanwhile Ferrier had also expressed a strong opinion adverse to the views of Wundt and Bain.[3] He said:—"But, whether we make the movements in reality, or revive them in idea, the consciousness of the extent and energy of the move-

[1] In regard to this point, see what I have said on p. 5 (Note [2]) of this paper.
[2] 'The Brain as an Organ of Mind,' pp. 540–544.
[3] 'Functions of the Brain,' 1st ed., 1876, p. 226.

ment is, in my opinion, in all cases dependent on in-going or centripetal impressions."

A few weeks after the publication of my work 'The Brain as an Organ of Mind,' there appeared a remarkable paper[1] by Dr. William James, of Harvard University, on 'The Feeling of Effort,' in which will be found many views strikingly similar to those that I have expressed. In reply to the question as to the nature of our sensible perceptions of movement he says:— "I unhesitatingly answer: an aggregate of afferent feelings, coming primarily from the contraction of muscles, the stretching of tendons, ligaments and skin, and the rubbing and pressing of joints; and secondarily, from the eye, the ear, the skin, nose or palate, any or all of which may be indirectly affected by the movement as it takes place in another part of the body. The only idea of a movement which we *can* possess is composed of images of these its afferent effects." Again he says:— "The *degree of strength* of our muscular contractions is completely revealed to us by afferent feelings coming from the muscles themselves and their insertions, from the vicinity of the joints, and from the general fixation of the larynx, chest, face and body, in the phenomena of effort, objectively considered. When a certain degree of energy of contraction rather than another is thought of by us, this complex aggregate of afferent feelings, forming the material of our thought, renders absolutely precise and distinctive our mental image of the exact strength of movement to be made, and the exact amount of resistance to be overcome."

Such language may seem very positive, but in my opinion also this second view as to the afferent nature of all the impressions by which we obtain a knowledge of our movements and by which we judge of weight and resistance is one that may now be considered to be incontestably established. Sachs[2] has shown the existence of sensory nerves in muscles, and such clinical evidence as is afforded by the cases of Demeaux, Landry, Spaeth, Duchenne, Briquet, and Bazire, show conclusively what opinion we ought to adopt in regard to this much disputed point. We have to do in these patients with

[1] 'Anniversary Memoirs of the Boston Society of Natural History,' 1880.
[2] Reichert's u. Du Bois-Reymond's 'Archiv.' 1874.

a total disappearance, in the parts affected, of all that kind of knowledge which has, by one or other, been ascribed to, or supposed to be derived from, the "muscular sense." They were ignorant of the position of their limbs, unconscious of any movements that they might execute, and of the degree of resistance which might be opposed to the contraction of these muscles. The volitional centres, the spinal motor centres, the motor nerves and the muscles were capable of being called into activity—yet all the information usually supposed to be derived through the "muscular sense" had vanished. No clearer evidence could be forthcoming to show that the knowledge of the position of our limbs, of their movements, and of the state and degree of contraction of our muscles generally, does not depend as Wundt, Bain, and others assume, upon impressions that are "concomitants of," or that coincide with, "the outgoing stream of nervous energy."

I go even further, and maintain that the processes taking place in motor centres and in motor nerves are purely physiological processes, wholly devoid of any conscious accompaniment—that is to say, from the time the motor incitation leaves the kinæsthetic centres, up to the time when muscular contractions occur, we have only to do with a series of physical processes occurring in different motor centres and in motor nerves.

Writing on this subject in 1880 I said[1]:—"The kinæsthetic centre is indeed one of great importance. Its impressions enter inextricably into a large number of our mental processes —as widely and inextricably, in fact, as the assumed muscular consciousness of Bain is supposed by him and others to be intertwined with what they would distinguish as passive sensibilities. But it can only produce an extreme amount of confusion if the activity of this sensory centre is attributed to and confounded with that of motor centres, the processes of which seem to lie even more truly outside the sphere of mind than the molecular processes comprised in the actual contraction of a muscle: these latter processes are, at least, immediately followed by 'ingoing' impressions, whilst so far as we know—that is, so far as any evidence exists—the former are

[1] 'The Brain as an Organ of Mind,' p. 600.

not." After so much as we are conscious of in the mere volition itself, "we have to do with molecular currents passing, it may be, through several sets of fibres and cells, but having no conscious side whatever." [1] Again, "Motor centres, wherever they may be situated, are parts whose activity appears to be wholly free from subjective concomitants. No ideal reproductions appear ever to take place in such centres; they are roused into activity by outgoing currents, and so far as we have any evidence, the induction in them of molecular currents which immediately afterwards issue through cranial and spinal motor nerves to muscles are simply physical phenomena." [2]

Views almost exactly similar to these were published only a few weeks later by Wm. James, in his essay on 'The Feeling of Effort.' Thus, he says (p. 20):—"In a word, volition is a psychic or moral fact pure and simple, and is absolutely completed when the *intention* or *consent* is there. The supervention of motion upon its completion is a supernumerary phenomenon belonging to the department of physiology exclusively, and depending on the organic structure and condition of executive ganglia, whose functioning is quite unconscious." Or again (p. 4):—"But apart from *à priori* postulates, and however strange to logic it may appear, it is a fact, that the motor apparatus is absolutely insentient in an afferent direction, although we know that the fibres of the anterior root will propagate a disturbance in that direction as well as in the other. Why may not this result from a true insentiency in the motor cell? an insentiency which would accompany all action there, and characterize its normal discharges as well as the unnatural irritations made by the knife of the surgeon or the electrodes of the physiologist upon the motor nerves." Again (p. 7)—"One immediate conclusion follows: namely, that there are no such things as afferent feelings, or feelings of innervation. These are wholly mythological entities. Whoever says that in raising his arm he is ignorant of how many muscles he contracts, in what order of sequence and in what degrees of intensity, expressly avows a colossal amount of unconsciousness of the processes of motor discharge."

[1] *Loc. cit.* p. 495. [2] *Loc. cit.* p. 599, see also p. 149.

I am happy to be able to cite another distinguished convert to these views. If any one will compare what was said by Prof. Ferrier in the first edition of his 'Functions of the Brain'[1] with what he says in the following passages taken from his recently-issued second edition (p. 436) it will be seen that he has made a most important renunciation, and now holds doctrines entirely in accordance with those above expressed. The views he now holds are these:—"The activity of the motor centres has no subjective side apart from the functioning of the sensory centres with which they are associated . . . whereas we have sensory ideation in and by itself, we have no ideas of movement apart from the sensory centres through which alone the activity of the motor centres is revealed in consciousness."

Many important issues in regard to cerebral localisation, as well as concerning psychological doctrine, turn upon the correct interpretation of the questions which we have just been considering.

In regard to the former, with which we are here chiefly concerned, it may be well to point out in the first place, that the interpretation of aphasia, aphemia, and all the varied amnesic states must differ widely from one another according to the different views which are taken as to the nature and seat of so-called "motor-ideas." The very notion of such a thing as a "motor-idea" could only arise in the mind of a man who believed we had feelings which could be felt, and

[1] At p. 266, he said:—"In the same manner as the sensory centres form the organic basis of the memory of sensory impressions, and the seat of their representation or revival in idea, so the motor centres of the hemispheres, besides being the centres of differentiated movements are also the organic basis of the memory of the corresponding movements, and the seat of their re-execution or ideal reproduction. We have thus a sensory memory and a motor memory, sensory ideas and motor ideas; sensory ideas being revived sensations, motor ideas being revived or ideal movements." Again at p. 271, he said:—"If the motor centres of the cerebral hemispheres are not merely the centres of impulse of volitional movements, but also the centres of registration and revivability of the same, it must follow that destruction of the cortical motor centres will cause not only objective motor paralysis, but subjective motor paralysis, or, in other words, paralysis of motor ideation." These views which Ferrier has, I am glad to say, renounced are thoroughly in accordance with doctrines still held by Hughlings-Jackson, and Stricker.

consequently ideas that could be revived, in motor centres. It so happens also that those who hold this doctrine have always been inclined to believe (though it does not of necessity follow), that such revived "motor ideas" constitute the material of our recollection when words recur to the mind in silent thought; this, for instance, has been the doctrine of Bain, Wundt, Hughlings-Jackson, and more recently of Stricker. Those, however, who repudiate the notion of the existence of any such things as "motor ideas," and who believe that all our ideas of movement are revived in sensory centres, have also for the most part supposed, that words are primarily revived in silent thought in the auditory centres, and that the kinæsthetic ideas of words are awakened in immediate succession thereto.

These divergent views lead to widely different interpretations of the various species of speech defects, and such interpretations entail quite different views as to the localisation of the lesions by which they are severally caused. This any one may see who will take the trouble to compare Stricker's account of such defects, as embodied in his work[1] 'Du langage et de la musique,' with that which I have advanced in a recently published work.[2] The different interpretations we have offered of so-called "word-blindness" and "word-deafness" might be looked to as an example of what I mean. The whole question is, however, far too large a one to be entered upon here.

Again, if our sensations of movement were "concomitants of the outgoing current," and if we really had "motor ideas" as well as sensory ideas, it would be perfectly natural and logical to look for motor centres in the cerebral cortex; but if these views are erroneous, and if all our knowledge of movements comes to us through sensory centres, then there is no longer the least ground for postulating the existence of motor centres in the cortex; all that would be needed there would be the existence of sensory registers of the impressions occasioned by movement. In other words, kinæsthetic centres clearly must exist in the cerebral cortex, to be as it were on the same platform with the registers for other sensory impressions with

[1] Paris (Biblioth. de philosoph. contemp.), 1885.

[2] 'Paralyses: Cerebral, Bulbar, and Spinal,' 1886, pp. 99-130.

which they are in such intimate functional relationship; whilst the actual need for the existence of motor centres in this situation has not yet been shown by any one.

The Part taken by "Muscular Sense" Impressions in the Execution of Voluntary and Automatic Movements.

(*a*) *Voluntary Movements.*

In voluntary acts we have not merely to account for the production of a movement, but of a movement of a certain kind, in which the action of each set of muscles brought into play is duly regulated so as to lead to the exact result desired. And, seeing that the volition or desire to bring about such and such movements originates in the cerebral cortex, so we are bound to admit, that the purely volitional qualities of the movement must also depend upon cerebral influence. Hence the strength, the continuance, the rapidity, and the direction of movements are variable according to the precise nature of the cerebral incitation or volition. As Jaccoud says, "The strength of the movement is regulated by the strength of the initial motor impulse; the extent depends in reality upon the same influence; the rapidity results from the more or less rapid succession of voluntary impulses; and the direction is determined by the voluntary localisation of the incitation upon certain groups of muscles." It is also important to recollect that, in the execution of a complex movement, any alteration that we may desire to bring about in respect of any one of these volitional qualities, is, by the mere change in the volition itself, immediately effected with reference to the movement as a complex whole. Jaccoud illustrates this as follows[1]:—"I am walking, and then I wish to walk more quickly. Hardly have I conceived the desire before the mode of walking is changed; it has become in short, more rapid. This intervention of my will manifests itself, therefore, by a change in the movement of locomotion as a whole. This change is the final result of a series of partial modifications which have been brought about in the original movement; this is incontestable. But I have

[1] 'Les paraplégies et l'ataxie du mouvement,' 1864, p. 594.

not needed a parallel series of volitional acts; a single volition has sufficed. I have willed to walk more quickly, and I have then walked more quickly, without knowing anything, without even requiring to know anything, of these intermediate modifications." In the same way, alterations in any one of the other volitional qualities of a complex movement are found to have direct bearings upon the movement as a whole. These seem to be the principal facts that should be mentioned with regard to the mere volitional act; and I am quite disposed to agree with Landry when he says, in opposition to the views of several physiologists previously mentioned, that the volition itself includes only an incitation to a specific kind of movement, and that we must execute this movement in order to become acquainted (inferentially) with the quantity of nervous action brought into play.

But besides these *qualities* of the movement which are determined by the volition, we have to consider the execution of the movement itself as a compound of several simple movements, brought about by the simultaneous and successive contractions of different muscles, which are perfectly harmonious and constant in their mode of action. This machine-like precision of action—the result of what is usually known by the name of *co-ordination*—is now admitted by most physiologists to depend upon certain pre-established, though gradually acquired, nerve-connections between the different elements of the spinal cord and medulla oblongata. The movements are machine-like, inasmuch as they depend upon certain organic combinations in the spinal cord and medulla, and after these connections have once been fully formed the will has little more to do with them; the movements ordinarily take place in a definite manner, the will qualifying their mode of execution only. It requires a special exercise of mental power in order to execute certain complex movements in a way different from that which has become habitual. But in these cases, as well as when we are learning to execute a new complex movement, we give no attention whatever to the muscles by which the movement is effected—the states of these individually do not fall within the scope of our consciousness. Were it otherwise, the number and situations of the several muscles would reveal

themselves to all, during the performance of different movements. In our tentative efforts we think only of the movements themselves—how to combine the more simple, so as to produce the more complex.

The kind of movement produced is, therefore, evidently dependent upon the *distribution*, in the medulla and spinal cord, of the volitional impulse; and, in a complicated motor act, its incidence upon particular groups of cells, which are the organic representatives of certain potentialities for simple movements, gives rise to the production of the complex movement. The more frequently such a complex movement has been executed, the more completely may we suppose these various groups of cells become bound together into one system by connecting fibres, and the more possible is it for the movement which they represent to occur in a thoroughly automatic manner, and often without much need for cerebral intervention. The mechanism of co-ordination is, therefore, purely spinal. This fact was fully recognised by Volkmann more than forty years ago, as appears from the following quotation:[1] "The physiological accomplishment of movements has nothing to do either with consciousness or with unconsciousness, for the mind has not the least notion of the details of this operation; and even in voluntary movements it knows nothing concerning the nerves or the muscles by the intervention of which the process is accomplished. In fact, the mind does nothing in this case but give the incitation; and if this incitation has for an effect any co-ordinated movement, it is altogether simply because the organ which receives the incitation is arranged in such a fashion that it necessarily produces a co-ordinated movement." Precisely the same views as these were also expressed, at about the same time, by Arnold and Müller.

Thus, the work of co-ordination tends to become entirely spinal and organic; the movement itself depends upon the spinal cord, though its particular *qualities* of force, rapidity, etc., are dependent upon the cerebral or volitional influence. The cerebrum may, therefore, also be said to exercise a kind of co-ordination—it co-ordinates or adapts the movements which are

[1] Art. 'Nervenphysiologie,' in Wagner's 'Handwörterbuch,' 1844.

organically represented in the spinal cord, so as to make them accord qualitatively with the aim conceived. But, in order that the cerebrum may exercise this power, it seems perfectly obvious that it should be instructed from moment to moment as to the exact nature of the movement actually produced, so that it may know whether to continue in its present mode of action, or whether to vary the quality of the volition, in order better to attain the desired end. Now, according to Jaccoud, whose treatment of this subject is most excellent, the cerebrum obtains these necessary guiding impressions from different sources. He says: "These indispensable notions the brain obtains directly through the sense of sight; or, instead it deduces them indirectly from the instructions which reach it as to the situation of the parts which move and the condition of the contractile organs which move them: these instructions are furnished by impressions through the muscular sense, and through the sense of touch." The sense of sight is what we chiefly rely upon in early years, and in acquiring new movements generally. We all know how long it is before a person learning to play upon a musical instrument can do without the aid of this guiding sense. At last, however, his tactile sense, and also his "muscular sense," has become so educated that he is able to do without guidance from the sense of sight; and, as a rule, he does without this primary aid as soon as the execution of the movement has become perfectly easy. Jaccoud calls the second mode of appreciation indirect, because it does not, as in the case of the sense of sight, depend upon a simple perception of transmitted sensations, but upon an interpretation of sensations. As he says[1]—"The sensorium requires, as a preliminary, to have learned the relations which unite the various conditions of the muscles, or of the tactile organs to the different sensations perceived: it is only at the termination of this apprenticeship that it can conclude from the sensation perceived as to the statical or dynamical conditions of the parts whence the sensation springs. This education proceeds correctly, by means of the direct appreciation through the sense of sight, thanks to which the individual can compare at each instant the movement effected with the sensation perceived."

[1] *Loc. cit.* p. 601.

After this preliminary education has been finished, the knowledge so acquired, though inferential, becomes as available and as efficacious as that which is more directly derived through the sense of sight. It can be brought into action also with just as much rapidity and exactness, so that the sense of sight is no longer needed to inform us as to the position of our limbs, and as to the nature and degree of their movements.

Jaccoud, therefore, thinks that the power which healthy persons enjoy of performing, with facility, movements that are at the same time habitual and complex depends, in the first place, upon the integrity of their "muscular sense," and in the second, upon that of their sense of touch. Impressions are derived through both these channels, whereby the brain becomes informed as to the amount and kind of movement which it has called into action; and so it learns whether, to obtain the end in view, it should continue with the same kind of volition, or whether it should be qualified in any way.

When we commence a movement, we initiate it with certain predetermined qualities of force and extent; and this, of course, is simply a result of our past experience and education. I know that certain objects have hitherto given me certain impressions of weight when I have previously handled them, and therefore my previous education now enables me, when I see such an object again and desire to handle it, to give the volitional act its necessary qualifications. This power has been termed "*l'instinct locomoteur*," and "*conscience musculaire*." It has been made the subject of much mystification, though it is a simple result of our capability of recalling in idea the kinæsthetic impressions which have previously been associated with given sight impressions; that is, the sight impressions immediately recall their related kinæsthetic impressions. When I see a simple bundle of wool on a table, as a result of previous experience I can at once nearly accurately determine what ought to be the quality of the volition necessary to enable me to raise it. Thus I am enabled to initiate such a movement as I deem appropriate. But supposing, in the case just cited, that the supposed simple bundle of wool was not a simple bundle, that it contained a heavy leaden weight in its centre, then my initial volition would have been inadequate, I should have

been deceived, and the kinæsthetic impressions that I received would have instructed me that a stronger volitional effort was necessary. It is always in this way that kinæsthetic impressions are supposed to intervene.

By a supposed "locomotive instinct" (or, in other words, ideal recall of kinæsthetic impressions), we know, as Jaccoud puts it, *what force we ought to employ*, whilst by the actual occurrence of kinæsthetic impressions we are taught *what force we have employed.*

I have now for some time advocated such views as these, and have maintained that the immediate execution of voluntary acts, in the case of the majority of limb-movements, is dependent upon the guidance of co-active visual and kinæsthetic centres; just as in the case of the complex movements concerned in articulate speech, the immediate execution of such movements is dependent upon the regulative activity of combined auditory and kinæsthetic centres.[1] The latter movements seem, in short, to be related to the auditory centres, in just the same sort of way that movements of the limbs are related to the visual centres.

Where the movements which it is desired to execute are complex and difficult and we have to learn them by imitation of the movements of other persons, the sense of sight is doubly brought into action. It is necessary at the commencement and during the continuance of our efforts to copy such movements, to look alternately at our model and at our own moving members. A long time and much practice is, in fact, required before a person who is learning to play upon some musical instrument, is able to execute the necessary actions without the aid, from moment to moment, of guiding visual impressions. During the process of learning, therefore, the visual centre evidently exercises a dominating influence.

In time, however, the impressions pertaining to the "sense of movement" (which are, of course, always associated with those of sight) become, by way of newly organized channels, sufficiently well associated with the newly organizing motor mechanisms, to permit the new movement whenever it has been initiated to be continued under the immediate guidance

[1] 'Brit. Med. Journal,' April, 1869, and 'The Brain as an Organ of Mind,' p. 555, and chap. xxix.

of kinæsthetic impressions only—that is, without further necessity for a conjoint direction through the sense of sight.

The same different tracts of the brain that are called into simultaneous or immediately successive activity for the initiation of any set of voluntary movements, would probably remain in activity during the continuance of such movement, though not exactly in the same relative proportions. Thus, if we suppose the centres specially called into activity, as guiding centres, to be the visual and kinæsthetic, it may well be that the former has a dominating influence in the production of the initial conception of the movement about to be executed. And yet the distinctness of this idea or conception of the movement (partly visual and partly kinæsthetic in its origin, as we have said) will be found to vary with the degree of familiarity, and consequently with the ease of execution, of the movement. In the case of the simplest voluntary movements, or those that have been often repeated, an idea or conception of the movement needed scarcely obtrudes itself at all as a conscious element of the volition. This is a part of the process which has here become more or less latent, and which in ideo-motor and sensori-motor actions has become wholly latent; though it probably still remains, even in these latter, as a necessary link in the chain of causation. On the other hand, during the continuance of voluntary as well as of almost all varieties of automatic movements, it seems clear that the kinæsthetic centres exercise the supreme guiding influence. Its impressions alone—even when they very imperfectly, or not at all, rouse our consciousness as to their existence—suffice to inform us (that is, suffice to excite their proper cerebral "centres" in ways definitely related to different positions and muscular tensions) as to the exact relations of our limbs, and as to the nature and degree of their movements.

The mode of acquisition above indicated, seems well to accord with our other interests and with the daily necessities of our lives. The sense of sight greatly facilitates the process of learning, and its vivid impressions speedily enable the brain to appreciate aright the more vague and occult impressions coming to it simultaneously through the kinæsthetic centres. Soon, however, the visual sense, which we need for so many

other important purposes, no longer requires to be concentrated wholly on the performance of movements. Later still, our attention or consciousness becomes further freed from disturbing details connected with movements. The possibly conscious impressions pertaining to the "sense of movement" at last habitually pass unheeded, and then we come to be able to perform multitudes of daily actions under the guidance of mere "unconscious" kinæsthetic impressions.

Thus the working of the motor side of our complex nervous mechanism, even when it is concerned in executing the behests of will, proceeds so smoothly, and is practically so much unheeded, as to leave us free to follow up the threads of our conscious life unhindered by the multitudinous details pertaining to the varying states of innumerable muscles acting in ever changing combinations.

Thus from what has been said it seems clear that the performance of a voluntary act is always preceded by an idea or conception of the movement we desire to execute; and that this idea or conception is, for ordinary movements, compounded of two kinds of past impressions, namely, those of the visual sense and those of the kinæsthetic sense. Again, it must be remembered that the kinæsthetic sense includes two different sets of impressions; the one set (*a*) being conscious impressions derived from our moving members (proceeding from skin, muscles, tendons, and joints); the other being (*b*) either unfelt, or almost completely unfelt, impressions emanating from the muscles, in relation with their varying states of contraction, and therefore affording information to the brain of the most important kind. The evidence demonstrating the existence of this last set of comparatively unfelt impressions emanating from muscles, and pointing to their importance for the guidance of movements, is mainly derived from cases of diseases such as have been already cited (pp. 10–20).

Now, in the chapter on "The Will," in his "Analysis of the Human Mind," published more than fifty years ago, it appears that James Mill clearly appreciated the fact, that the "idea of the action" to be performed is two-fold. He says, "There are two ideas very different from one another, to both

of which we give the name 'idea of the action.'" Of these, he adds, "one is the outward appearance of the action, and is always a very obvious idea." The other is a copy of certain internal sensations, which a few pages before he had spoken of generally as sensations accompanying the movement, and which he also more specifically defined (*loc. cit.* p. 275) when speaking of the terminal events of a movement as "the contraction of the muscles, with the various sensations which the action upon those organs, and the action excited in them, imply." Of these internal sensations he says, "from the habit of not attending to them, we have lost the power of attending." And then he adds, "This last (namely the revival of such internal sensations) is by no means an obvious idea. And the mind passes from it so quickly, intent upon the action which is its result, that it is almost always swallowed up in the mass of association. It constitutes, in fact, one of the most remarkable instances of that class of links in a chain, which, how important soever to the existence of the chain, are passed over so rapidly, that the existence of them is hardly ever recognized. . . . *This last idea alone is that upon which the contraction is consequent.*"

This view is then essentially that which I myself adopt, viz., that kinæsthetic impressions, and especially those of which we are least conscious, are the last to be revived in the cerebral cortex, anterior to, and as actual last links in the chain of cerebral processes which determine, the excitation of the motor centres themselves.

I find that since the first publication of these views, very similar notions as to the importance of revived sensations of movement for the execution of voluntary acts have been expressed by two other writers.

The views of one of these, namely Dr. E. Fournié, were published three years afterwards;[1] and though we do not at all agree in our general interpretation of perceptive processes, nor as to the regions of the brain concerned therewith, we are thoroughly in accord as to the importance of these sensations of movement in conjunction with other sensory impressions as guides for movements generally. Thus, he fully

[1] 'Physiolog. du syst. nerveux,' 1872, pp. 291, 360, 362–5, 447

recognized the important part played by revived sensations of muscular contraction in voluntary as well as in instinctive movements; also the difficulty of reviving such impressions alone, when they have not been linked with visual or auditory impressions. It is true, he does not seem clear as to the mode in which such revived sensations of movement come into play, though he firmly believes that conjoined sensory impressions are the true cerebral co-ordinators of movement, and that by the aid of these alone we are enabled to learn new movements. He holds that sensations telling of the state of contraction of the muscles are "indispensable conditions in order that each movement may be directed in a suitable fashion," and says that, but for our power of recalling in idea or reviving such impressions, new movements could never be perfectly learned, so that the "execution of movements would be an eternal apprenticeship."

But much more important and more generally in accordance with my own views are the statements made by Dr. W. James. He says [1]:—"The essentials of a voluntary movement are—(1) a preliminary idea of the end we wish to obtain; (2) a '*fiat*'; (3) an appropriate muscular contraction; (4) the end felt as actually accomplished. In man at any rate it is admitted that the idea of the end and the muscular contraction were originally coupled by empirical association; that is to say, the child with his end in view, made random movements till he accidentally found one to fit. This movement awakened its own characteristic feeling which thenceforward remained with him as the idea of the movement appropriate to that particular end.[2] If the man should acquire a million distinct ends, he must acquire a million such motor ideas and a million connections between them and the ends. But one such connection, subserved by an exclusive nerve tract used for no other purpose, will be enough for each end. The end conceived, will, when these associations are formed, always

[1] *Loc. cit.*, 1880, p. 5.

[2] In a note further on, Dr. James calls attention to an important point. He writes (*loc. cit.*, p. 9):—"I may add that in teaching a new and unnatural movement, the starting-point is to awaken by its passive production a distinct sense of what the movement, if effected, would feel like. This defines the direction of the exertion the pupil is to make."

awaken its own proper motor idea. As for the manner in which this idea awakens its own proper movement—the one which will convert it from an idea into an actual sensation—the simplest possible arrangement would be to let it serve directly (through its peculiar neural process) as a stimulus to the special motor centre, the ultimate sensible effect of whose discharge it prefigures and represents." Again, he says:—"If I will to write 'Peter' rather than Paul, it is the thought of certain digital sensations, of certain alphabetic sounds, of certain appearances on the paper, and of no others, which immediately precedes the motion of my pen. If I will to utter the word 'Paul' rather than Peter, it is the thought of my voice falling on my ear, and of certain muscular feelings in tongue, lips, and larynx, which guide the utterance. All these feelings are afferent, and between the thought of them, by which the act is mentally specified with all possible completeness, and the act itself, there is no room for any third order of mental phenomena. Except indeed what I have called the fiat, the element of consent, or resolve that the act shall ensue. This, doubtless, to the reader's mind, as to my own, constitutes the essence of the voluntariness of the act."[1]

But the so-called "fiat" has no particular mystery about it. It is the mere result of the fact, that one out of the two or more motives which weigh themselves against one another, is, after little or much deliberation, recognised to be stronger than the others; the result being that the molecular movements with which it is associated are permitted to flow over into motor channels so as to evoke the fitting muscular actions. As James says:—"In our bed we think of the cold, and we feel the warmth and lie still, but we all the time feel that we can

[1] In regard to the nature of this "fiat" we may say, with James Mill, that it is simply equivalent to a *desire* sufficiently strong to be immediately operative (see 'The Brain as an Organ of Mind,' p. 550). Hartley said also, "The Will is, therefore, that desire or aversion which is strongest for the present time." Which mental mood is to prevail is sometimes immediately settled, and at other times only after a process of Deliberation. Concerning this process Hobbes said:—"The whole sum of desires, aversions, hopes and fears, continued till the thing be either done or thought impossible, is what we call *Deliberation*. . . . Appetite, therefore, and aversion are simply so called as long as they follow not deliberation. But if deliberation have gone before, then the last act of it, if it be appetite, is called *will*; if aversion, *unwillingness*."

get up *if we will*. The difficulty is to will." In the case of emotional actions, the mere presence of the exciting idea is much more apt immediately to excite the corresponding movement, so that "the discharge of idea into movement is much more readily inhibited by other casually present ideas in the case of voluntary action, and less so in the case of emotions; though here too inhibition takes place on a large scale." As a result of these considerations he lays down the following important conclusion,[1] "*that every representation of a motion awakens the actual motion which is its object, unless inhibited by some antagonistic representation simultaneously present to the mind.*"

W. James very neatly summarizes his own as well as my views on this part of the question, when he says:—"The ordinary 'voluntary' act results in this way: First, some movement produces a feeling in a reflex, or as we say, accidental way. The movement excites a sensorial tract, causing a feeling which, whenever the sensorial tract functions again, revives as an idea. Now the sensorial and motor tracts, thus associated in their actions, remain associated for ever afterwards; and as the motor originally aroused the sensory, so the sensory may now arouse the motor (provided no outlying ideational tracts in connection with it prevent it from so doing). Voluntary acts are, in fact, nothing but acts whose motor centres are so constituted that they can be roused by these sensorial centres, whose excitement was originally their effect."

Finally, it would seem clearly to follow, from the views above set forth, that in all essential respects the cerebral mechanisms for the actual production of voluntary, of ideo-motor, and of emotional movements are identical—that is to say, that the "way out" from the sensory centres in which the idea of the movement to be effected is revived, and the efferent tracts thence onwards towards the motor centres by which the movement is to be actually evoked are, when the movement itself is similar, identically the same in each of these cases.

The sensory components of the idea of the action about to be performed are, moreover, always twofold. They either belong to the visual and the kinæsthetic orders, or else they are auditory and kinæsthetic, as in the case where speech movements are to be evoked. The cases are rare in which, with ordinary seeing

[1] *Loc. cit.* p. 17.

individuals, we have the combination in idea of special tactile with kinæsthetic impressions. In the blind, however, this particular combination for the ideal recall of movements becomes very common.

In each case the "conception of the movement" about to be executed, being a means rather than an end, does not after a time attract our attention. If this is so even with the initial guides of movement (that is, the revived visual and auditory impressions), how much more may we expect it to be the case with the revived kinæsthetic impressions, which are always less vivid. And how little even the initial guides of movement, when revived in idea, at all clearly reveal themselves to consciousness in many persons is evidenced by the fact, that it is denied by Prof. Stricker and some other persons that words are primarily revived, anterior to articulation, in the auditory centres.

It is, however, neither customary nor needful either for thought or for action, that the several links in the chain of association by which these processes are effected should reveal themselves in consciousness. Many of the links of our thought and of our sensorial action are represented by sub-conscious nerve processes, and this is clearly the case with regard to those linked revivals of sensory activity which immediately precede and guide muscular movements. Transitions from conscious to unconscious nerve actions are habitually taking place during the education of the individual and the development of the nervous system in each one of us; and yet the essential nerve actions either still go on in the latter case, and none the less surely exercise their influence, in evoking other nerve actions which are associated with conscious states, or in leading to the production of this or that form of movement.

(b) *Automatic Movements.*

As we have seen, between voluntary and ideo-motor or emotional acts no really intrinsic differences exist, from the point of view of the nerve centres concerned with their production.[1]

[1] In the first edition of his 'Functions of the Brain,' Dr Ferrier distinctly argued as though the nerve mechanisms needful for the production of voluntary

Are we to hold, however, with Ferrier and others, that there are special motor centres for the performance of voluntary actions altogether apart from those by the aid of which automatic actions are performed? This is a view to which I cannot subscribe. An automatic movement is simply what was once a voluntary movement in the race from which the individual has sprung. The motor mechanisms that are now immediately concerned with its execution are precisely those which were originally linked together or "organized" when such a movement first began to be effected by voluntary efforts.

The difference here, as I contend, is not with the motor centres; it lies rather with the nervous processes which precede the action of the motor centres. Thus, when eating, one swallows food by a series of automatic actions, without taking heed of the process; but I can at any time, by directing my attention thereto, and with the presence of saliva as an additional stimulus, voluntarily perform these same processes. In the one case the movements are evoked as mere reflex actions in which the medulla is the centre concerned; in the other they are evoked under the influence of volitional stimuli whose birth and starting-point is to be found in the cerebral cortex.

It is, therefore, in my opinion, a fundamental error to look for special motor centres for the production of voluntary movements of any kind—either in the cortex or elsewhere.

The cerebral hemispheres are needed for the learning of new movements which in succeeding generations of animals come to be performed in a purely automatic manner; or, we ourselves may learn movements which ultimately by dint of time and long practice may become so facile that they take their place in the category of "secondary-automatic" actions. These facts seem to me to show conclusively that, in the learning of any new movement, two processes of education (entailing the establishment of structural connections, or the laying down of new nerve routes) must take place concurrently.

movements were different from those by which ideo-motor and emotional movements are evoked. In the recently issued second edition he does not so explicitly state this view (*loc. cit.*, p. 371), nor does he clearly retract it.

One must be carried on in the cerebral cortex in the way already described; whilst another must be effected by developmental processes taking place in lower centres, of the action of which we are altogether unconscious—processes which, with lapse of time and successive generations, ultimately permit such movements to occur in a purely automatic fashion, and, it may be, without any concurrent stimulus either reaching or rousing the activity of the cerebral cortex.

It has now been perfectly well established, by experiments on lower animals, that even such complex muscular actions as are required for station and locomotion may be performed in pigeons and in rabbits after the cerebral hemispheres have been removed. Here, therefore, it is clear we have only to do with a series of very complex reflex actions, carried on by means of the mesencephalic and cerebellar centres. It is true that afferent impressions of various kinds are needed for the effective performance of the very complex muscular actions required for the maintenance of equilibrium and for locomotion—there must, in fact, be the advent of proper visual impressions, of labyrinthine impressions, of tactile impressions, and above all of "muscular sense" impressions. With the sole presence of these, however, the necessary movements for locomotion may be evoked, even though in its natural condition the animal is also in the habit of constantly inciting these same complex movements by volitional stimuli. The motor centres in action remain the same, they are merely called into play in a different manner; and in each case the actual movement, whether reflex or volitional, is evoked by stimuli starting from sensory centres.

For the execution of many other automatic movements the kinæsthetic impressions which impinge upon lower centres are of less importance than tactile impressions; these latter, in many cases, are the special stimuli which evoke the acts, whilst kinæsthetic impressions here also serve as absolutely unconscious guides—especially where the act is one which involves a succession of muscular movements. It must be upon the basis of information coming to the spinal cord itself in the form of kinæsthetic impressions, that, even in animals in which the cerebral hemispheres have been removed, as Goltz has shown,

we may at times observe new adaptive movements called forth of a distinctly purposive type, that is, with a view to remove some unusual stimulus. This is an adaptive power which, as Dr. Ferrier admits,[1] the evidence compels us to concede to the spinal cord.

This kind of use to which kinæsthetic impressions are put in the case of automatic movements, holds good for movements of different degrees of complexity, and equally so whether they are evoked under the guidance of mesencephalic, of medullary, or of spinal sensory nuclei.

But by the time a new movement has been thoroughly learned by any particular race of animals, that is, after its constant repetition by successive generations of such animals, these tactile and kinæsthetic impressions to lower centres (whether they be spinal, medullary or mesencephalic) having been habitually impinging upon such centres (as well as making their way to the cerebral cortex) must have opened up direct channels of communication between such lower sensory centres and the related motor mechanisms, so that the corresponding movements are then capable of being evoked in a purely reflex fashion, after the manner of automatic movements generally—that is, on the mere occurrence of the suitable stimulus, and without necessity for the intervention of the cerebral hemispheres. It is true that, in the majority of cases, the cortical connections with such motor centres still exist, so that the same movement may, whenever the desire is felt, continue to be evoked by a voluntary stimulus. This, as we have seen, is the condition of things with regard to the movements of deglutition already referred to, as well as for station and locomotion in many of the lower animals.

Experiments upon lower animals seem, indeed, to teach us that if we take different creatures such as frogs, rabbits, dogs,

[1] 'Functions of the Brain,' 2nd ed., p. 118, where he says:—"By a series of ingeniously contrived experiments Goltz has shown that, even when the limbs of a frog were so fixed or placed in positions which could not have occurred in its past experience, the animal, without its hemispheres, retained the power of adapting its movements in accordance with these unusual and abnormal conditions. This would indicate that, if these centres are merely centres of reflex action, the reaction is that of a machine possessing in some way the power of self adjustment."

or monkeys, we find in the first mentioned a comparatively small number of movements that are not purely automatic, and consequently a very small number of these which are permanently interfered with by a removal of their cerebral hemispheres. In rabbits and dogs, however, we find movements more and more interfered with after removal of the cerebral hemispheres, showing, according to my interpretation of the facts, that a larger proportion of the movements of these animals are as yet unemancipated from the necessity of cerebral control and guidance in the manner I have indicated through sensory centres. In monkeys, again, we meet with a large increase in the proportion of movements which are still so comparatively new (for the race), as to be only capable of being evoked by stimuli emanating from the cerebral cortex. While in man, the knowledge we have acquired from the study of disease, as well as our knowledge of the infinitely diversified uses to which the human hand may minister, suffices to convince us that the influence of the cerebral cortex in the production of movements even very greatly surpasses that which obtains with the monkey.

Thus, we find in man the kinæsthetic centres, as well as the visual, auditory, and tactile centres, alike obtaining their maximum development, because of the extremely diversified uses and the complexity of the processes to which they are called upon to minister. The very fact of the greater development of all these sensory centres in man, and of the enormous number of the movements which are suggested and guided by his intelligence, probably tends to maintain movements generally in him more, and for a longer time, under the influence and guidance of the cerebral cortex, than similar movements would be in lower animals. Hence the larger amount of motor defect produced in man by certain cortical lesions, simply because these lesions destroy those sensory centres in the cortex from which the volitional stimuli or controlling incitations immediately issue.

This is a kind of explanation which seems to me much more harmonious with all the facts than that advanced by Dr. Ferrier and others, which supposes that there are motor centres for voluntary movements altogether distinct from those for auto-

matic movements, and that these imaginary "motor centres for voluntary movements" are situated in the cerebral cortex.

The Cortical Localisation of "Muscular Sense" Impressions.

We have seen that there are practically three groups of cases in which loss or defect of "muscular sense" impressions is apt to be met with (p. 21) :—(*a*) cases where there is disease in the posterior columns or posterior cornua of the spinal cord; (*b*) cases of cerebral hemianæsthesia, in which there is functional or structural disease in the region of the posterior extremity of the internal capsule; and (*c*) cases which I previously spoke of as a more ill-defined group, but which I will now venture to say are cases of disease in the so-called motor regions of the cerebral cortex.

In regard to the precise path through the spinal cord taken by muscular sense impressions, nothing definite can as yet be said, beyond what has been above indicated. It is principally with some forms of locomotor ataxy, among spinal diseases, that defects of "muscular sense" are met with, therefore we suppose these impressions must pass upwards either through some part of the posterior column or else through the posterior cornua of the cord.

We are also unable definitely to trace the path pursued by "muscular sense" impressions through the medulla, pons, and cerebral peduncle. The fact, however, that in a very large proportion of cases of cerebral hemianæsthesia no loss of the muscular sense exists while, in other rare cases, these special impressions as well as any others that may be derived from muscles are entirely absent, tends to make it probable that in or near the posterior part of the internal capsule the channels for these impressions are gathered together in a more or less distinct fasciculus, before they diverge on their way to the cerebral cortex.

The final question that now presents itself for consideration, however, is :—What are the parts of the cortex to which these "muscular sense" impressions proceed? Can this be said to be known, or not?

In seeking to obtain an answer to this question, we ought to look for certain evidence that may be obtainable from experiments upon lower animals, and we should supplement this information by other evidence of a clinico-pathological nature derived from the effects of disease of the cerebral cortex in man, or from the effects resulting from removal of portions of the cortex on account of disease in this situation.

Evidence obtainable from lower animals.

What information may we fairly seek to obtain on this subject from experimentation upon the lower animals, and especially upon monkeys? From what has been said as to the functions of the "muscular sense" centres in the cortex in reference to movements generally, it seems obvious that they must be divided into two classes, from the point of view of the degree of their relationship to the activity of the cerebral cortex. Thus we have :—

Class I.—Voluntary actions in the largest sense of that term, among which we would include all those movements of varying degrees of facility (that is ranging from new difficult movements, to those of ideo-motor type) which cannot be executed apart from cortical instigation and guidance, proceeding from the opposite cerebral hemisphere.

Class II.—Actions that are only at times incited voluntarily (that is, by incitations from the cerebral cortex), but which are, in the great majority of cases, evoked by the reflex activity of lower centres as true automatic movements.

It is necessary to bear this distinction in mind because, as will be seen, the results of destruction of the "muscular sense" centres in the cerebral cortex on the one hand, and of their stimulation on the other, should be different for the two classes of movement.

Supposing, for the sake of our argument, we have *destroyed* the whole of the "muscular sense" centres of one hemisphere (the centres through which, in all actions that are performed voluntarily, the motor centres of the opposite side of the pons,

medulla, and cord, receive their ultimate guiding stimuli), the following result ought to ensue :—

	Classes of Movement.	Results.
Destruction of "Muscular Sense" Centres in One Hemisphere.	Movements of Class I. . .	All of them paralysed.
	Movements of Class II. . .	These movements not paralysed.

With practice under such conditions, and after some time, it is probable that some of the movements of Class I. which were nearest akin to those of Class II. would become capable of re-execution. They would pass indeed from the one class into the other—so that some amount of recovery from paralysis might be brought about in this way.

Now let us, in the same way, consider the effect of *stimulation* either of all or of some of the "muscular sense" centres in one hemisphere, and ascertain the effects that should be induced in regard to the same two classes of movement :—

	Classes of Movement.	Results.
Stimulation of "Muscular Sense" Centres in One Hemisphere.	Movements of Class I. . .	Contractions or spasms affecting the muscles by which these movements are performed.
	Movements of Class II. . .	Do. do.

Evidence obtainable from observations on Man.

From destruction or stimulation of the "muscular sense" centres in man, we may look for just the same kind of effects, in reference to the two classes of movement, as we have above said might be expected to occur in the lower animals. There should be a difference in degree only, dependent upon the fact of the far larger proportion of movements which, in man, belong to Class I., as compared with Class II. As a matter of fact (thanks in great part to the experimental investigations of Dr. Ferrier), we are now all familiar with the paralyses that result from destructive lesions in the Rolandic area of the cortex, and with the various forms of "Jacksonian Epilepsy" resulting from irritative disease in these same regions.

Other defects, however, may be looked for in the case of man, over and above those of which we can obtain distinct evidence from animals, as a result of destruction of his

"muscular sense" centres. These are certain defects of sensibility of such a kind that it would be quite hopeless to attempt to obtain any clear and reliable evidence concerning them by experiments upon, and observation of, any of the lower animals.

It is all the more necessary to insist upon this point, because some experimenters have drawn conclusions from their observations as though it were possible to obtain such evidence from the lower animals. But, I would ask, how is it possible for us to form any trustworthy judgment concerning a blindfolded animal's knowledge (1) of the extent of the active or passive movements of one of its limbs, or of the exact position in which it may be left at the termination of these active or passive movements; or how, again, (2) are we to judge whether the animal's ability to discriminate differences in weight or resistance with one of its limbs has suffered any diminution? These are much the most valuable means of testing the integrity of the "muscular sense," yet no evidence can be obtained in regard to either of them which is of the least value, except from an animal that has the power of speaking and telling us what his impressions are under the various trial circumstances. The only other kind of test (of less value than the two above-named, because it leads to more equivocal results) is (3) the testing of the animal's ability to perform certain definite movements with one of its limbs when its eyes are closed. How difficult it must be to obtain any reliable information upon such a subject by observations upon a blindfolded animal can be easily imagined, if we look only to the limited range of movements of a suitable kind which the animal could be induced to perform under such conditions. This, however, is not the whole extent of the difficulty that we should have to face in such an investigation; there is the further complication, that the animal's limb which it would be most important to test in regard to the integrity of its "muscular sense" impressions, in the particular experiments with which we are concerned, would be partially paralysed, and thus all attempts to test the integrity of its muscular sense impressions by this third and only means which is available in animals would be rendered still more fallacious and untrustworthy.

I am strongly of opinion, therefore, that all the statements which have been made in reference to the integrity or otherwise of special "muscular sense" impressions, in any of the lower animals that have been operated upon, are altogether valueless,[1] and that we must look only to observations upon man to throw any true light upon this side of the question.

The additional defects which ought to be met with in man, however, as a result of disease of the "muscular sense" centres are of this kind. Destructive disease, or removal by the surgeon, of parts of the "muscular sense" centres should lead, in proportion to the completeness of the destruction of the parts, to a more or less marked loss of that kind of knowledge which comes from the "muscular sense" in connection with the parts of the limb that are paralysed, or the whole limb or limbs, as the case may be. In a patient in whom ordinary tactile sensibility is not much affected, if we find, after various random passive movements with eyes closed, that he cannot tell correctly the position in which his limb or fingers may be left, the evidence becomes all the stronger that there must be a notable defect in the "muscular sense," because, as may be supposed, in such a case the patient may derive some guidance towards such knowledge through skin impressions. In these cases, therefore, the loss of the "muscular sense" would *always seem to be less than it really is*, and unless great care be taken it would be easy for a superficial observer to come to the conclusion, that it was not impaired at all. One precaution, for instance, should always be taken. That is, the observer, after passively moving the patient's limb or fingers, must be careful not to let his own fingers remain in contact with the patient at the end of these passive movements, when he asks as to the position in which the limb or fingers may have been left. If, however, we should have our suspicions that part of a patient's ability to tell the nature of passive movements made, or of the position in which his limb is left, comes from the guidance that he derives through the tactile sense, we must resort to other methods before we arrive at a positive opinion as to whether the "muscular sense" is defective or not.

[1] See note to p. 9.

In doubtful cases, therefore, perhaps the most trustworthy means of arriving at a positive conclusion as to the integrity or otherwise of this sense are these three (neither of which would be applicable, be it observed, in the case of one of the lower animals):—(*a*) Ascertaining whether the patient has or has not a complete knowledge of the position of the limb or limbs in question on first awaking from sleep, and before they are moved in any way; (*b*) Testing his ability to appreciate differences in weight with the part affected[1]; and finally (*c*) we may resort to a method adopted by Westphal and also by Horsley (a method which is particularly useful when the part to be tested is much paralysed), that is, ascertaining when the patient's eyes are closed with what readiness, or the reverse, he may be able to bring the forefinger of the sound hand at once upon any particular part that may be indicated to him (though not touched) of the affected limb in a state of rest. With a defective knowledge of the presence and position of a limb, the power of at once finding a particular part indicated would probably be distinctly impaired.

In brief, then, it comes to this, that if we have regard to the functions of the "muscular sense" department of the kinæsthetic centres (of which they form the nucleus or most essential part), we should expect that their stimulation would lead to spasms and convulsive movements of the parts whose motions are in relation therewith; that destruction of such centres would, on the other hand, lead to a paralysis of those movements which have hitherto only been evoked by the action of these centres; and, further, that in this latter case, there would also be a loss or marked diminution of "muscular sense" in the parts principally affected.

It will at once be seen that these are precisely the effects which have now been ascertained to follow upon the stimulation and destruction of the so-called "motor centres" in the cerebral cortex, the position of which, thanks to the successive labours of Fritsch and Hitzig, of Ferrier, of Carville and Duret, of Exner, of Horsley and Schäfer, and of Beevor and Horsley, have now been very thoroughly ascertained. The

[1] With specially charged gun cartridge cases after Galton's method, or some other equally delicate means. (See 'Inquiries into Human Faculty,' 1883, p. 370.)

position of the centres in connection with different movements of the limbs and trunk have now, in short, been definitely localised by these various workers as well as by clinical observers, in different parts of the Rolandic area and of the marginal convolution.

My opinion, therefore, is that the evidence in our possession points very strongly to the conclusion, that Ferrier's so-called "motor centres" are in reality kinæsthetic centres in which "muscular sense" impressions in particular have been registered. The following reasons lead me to this conclusion:—

1.—All the effects resulting from the stimulation or destruction of these centres are, as I have already shown, in accordance with this view.

2.—This being so, the view that "motor centres" exist in the cerebral cortex cannot be correct, unless it can be shown that there is in the cortex of each hemisphere another totally distinct set of centres, the stimulation of which evokes definite movements, and the destruction of which involves an inability to execute the same movements. But, both experimental physiology and clinical medicine speak strongly to the fact, that there is but one set of areas (Rolandic and marginal) in which irritation or destruction leads to any such results. I claim, therefore, that these areas must be in great part (whatever other functions they may discharge) devoted to the registration of kinæsthetic impressions of the "muscular sense" order, which, as I have endeavoured to show, are so all-important for the production of voluntary movements.

3.—No valid reasons have ever been brought forward against this view. To this it may be replied that Dr. Ferrier has carefully examined such doctrines, in the last edition of his 'Functions of the Brain' (pp. 379–381), and that he has there shown how much such views are at variance with existing knowledge.

The necessities of the case compel me, therefore, to be frank concerning Dr. Ferrier's position in regard to, and his treatment of, this question, to which I have given considerable attention.

According to Hitzig[1] the so-called motor centres are the

[1] Reichert's u. Dubois-Reymond's 'Archiv,' 1870 and 1874; also 'Untersuch. in das Gehirn,' p. 59, 1872.

centres for the "muscular sense" or "muscle-consciousness," while according to Nothnagel,[1] though not the centres for, these regions are traversed by, impressions of this order. Both, however, adopt that lax view of the "muscular sense" which would make it include impressions from joints, skin, etc., or, in other words, all the impressions resulting from movement, which I have grouped together as kinæsthetic impressions. Thus their views, although nominally the same as mine, are really different, seeing that I am content to subscribe to the views of Ferrier as well as of Horsley and Schäfer, to the effect that the falciform lobe (hippocampus, gyrus hippocampus, and gyrus fornicatus) is the main seat, at all events, in which tactile impressions and those of common sensibility are registered. The convolutions in the excitable Rolandic area together with the marginal convolution, are, however, according to my view, the seats in which "muscular sense" impressions proper (the all-important constituents of kinæsthetic impressions) are registered, and from which volitional stimuli immediately issue. Arguments, therefore, which may be valid against the views of Nothnagel and Hitzig, in regard to the non-impairment of common sensibility in cases of destructive disease of the parts in question, may be of no avail against my doctrine.

As, however, we have much in common, it may be well to scrutinise Dr. Ferrier's attitude with regard to these views as a whole, especially as he does not endeavour to make any nice or essential distinctions between them.

Thus, he does not attempt to distinguish between the effects that might be expected to follow from loss of the "muscular sense" centres, and those which would result merely from the cutting off of the proper impressions from such centres, they themselves being left intact. Any one who has read the present paper will realize that from my point of view this is a most important distinction. In his first edition Ferrier pointed out that Nothnagel regards the cortical centres in question as "in some manner directly connected with the paths" of muscular sense impressions, while Hitzig regards them as the cortical termini for these impressions. In his second edition, he does not think

[1] Virchow's 'Archiv,' Bd. lvii. 1873.

it worth while to state any such distinction; he assimilates Nothnagel's doctrine to that of Hitzig, and, strange to say, assumes that the effect of the destruction of such centres would be ataxy rather than paralysis. He even repeats this as against my doctrine, saying, "It might explain ataxy, but not paralysis." This, as well as other remarks which he makes, convinces me that Dr. Ferrier has given no adequate consideration to the subject. Let me say definitely that, in accordance with my views, paralysis should result from the destruction of these centres; and that inaccurate and more or less disordered movements when the eyes are closed (ataxy) should be the effect of the cutting across of the paths of muscular sense impressions whether near the hinder part of the internal capsule or in the spinal cord.[1]

Ferrier, treating the question from the point of view of the mere cutting off of muscular sense impressions (which is in fact Nothnagel's doctrine, though not Hitzig's which he is ostensibly criticising), supposes, therefore, that the resulting defects would be of an ataxic order more especially, and then goes on to say that this is, at all events, not the nature of the defect met with in the monkey or in man. Then comes the following very positive statement:—"It is also certain—and a subject of daily clinical demonstration—that in paralysis from cortical disease the patient though unable to move his arm voluntarily, is perfectly aware of every movement passively communicated to it, and can state with exactitude whether his arm is flexed or extended, whether his fist is closed or open, and whether his finger is being flexed or extended gently or with force. His muscular sense, as well as every other form of common sensibility, is absolutely unimpaired." How very far this is from being an accurate statement, however, any one may judge

[1] In relation with this statement, it may be well here to recall the fact, that if a low condition of functional activity in the "muscular sense" centres should co-exist with a disease which cuts off its proper impressions from such centres, there may, in addition, be inability to perform the simplest movements when the eyes are closed (pp. 16 20 and 36); while, in other cases, with a still lower state of functional activity of the "muscular sense" centres (with or without co-existing hemianaesthesia), we should have to do with paralysis of a *hysterical type*, in which voluntary movements cannot be performed either with eyes closed or with eyes open (p. 36).

who will refer to the cases which I have previously cited (pp. 24–32), viz. the case of P. Zenner, presumably one of cortical injury, as well as the two cases recorded by V. Horsley, in which he had excised portions of the cortex. Afterwards, in his attempts to dispose of the doctrines of Hitzig and Nothnagel, Dr. Ferrier makes a series of statements which, to say the least, are, as I have attempted to show in previous parts of this paper, very insufficiently founded upon fact and actually misleading in their nature.[1]

In short, it seems to me perfectly obvious that Dr. Ferrier has never thoroughly thought out this portion of his subject, and that his expressed opinions have been, and still are, most inconsistent. He began, in the first edition of his justly celebrated work, by assuming that motor centres were seats in which the ideal recall of movements occurred[2]—though at the same time, and in flagrant opposition therewith, he strongly contended that all sensations resulting from movement reach the brain through afferent channels.[3] In the second edition of his work he has corrected this discrepancy. He now admits that the ideal recall of movements can only occur in sensory centres—though he appears to do all he can to explain away the importance of these kinæsthetic centres. He shows no gleam of recognition of their importance for the execution of voluntary movements.[4] He, indeed, expressly repudiates their influence in this direction (and the opinions of those who support such a notion) principally by means of two assertions which are almost wholly at variance with clinical teaching. Thus, he makes the extraordinary statement, that loss of tactile sensibility carries with it loss of the muscular sense;[5] and, further, that loss of the muscular sense never occurs alone, or, as he implies, otherwise than in direct proportion to loss of tactile sensibility.[6]

Dr. Ferrier's statements concerning the order of events in voluntary acts are, moreover, extremely inexplicit; his assumption that the mechanisms for such acts are organised in

[1] I mean the statements which I have quoted in Notes to pp. 15 and 23.

[2] See (*loc. cit.*) the first paragraphs of § 92, § 96, and § 98.

[3] *Loc. cit.* pp. 218–228.

[4] *Loc. cit.*, 2nd ed., pp. 432–438.

[5] See note on p. 22 of this paper.

[6] See note on p. 23 of this paper.

special cortical centres, rests on just as unsatisfactory a basis as does his notion, that all kinæsthetic impressions are mixed inextricably with tactile impressions in one and the same cortical terminus.[1] As this, in fact, is a rival hypothesis, I ought to quote it in Dr. Ferrier's own words, more especially as it is all, so far as I have been able to find, that he says throughout his work and on his own account concerning the cortical localisation of "muscular sense" impressions. After remarking that he considers it established "beyond all doubt that the falciform lobe is the centre of common and tactile sensibility," he adds[2]:—"In the same regions are also the centres of cutaneo-mucous and so-called muscular sensibility. . . . All the facts receive the most satisfactory explanation, if we regard the falciform lobe as a whole, and in each and every part the centre of tactile sensation for the whole of the opposite side of the body; though probably the various motor centres are each anatomically related by associating fibres with corresponding regions of the falciform lobe. This association would form the basis of a musculo-sensory localisation."

In the few remarks which Dr. Ferrier has made in regard to my views in particular,[3] he principally confines himself to a statement of the difficulties which he experienced in comprehending their precise meaning. These difficulties I trust he will now no longer experience; at least I have striven to explain my meaning as clearly as possible. Several of his statements concerning these views are not altogether correct, but one which he makes is extremely misleading, in more ways than one, and calls for some notice from me. He leads his readers to believe, that my views should not find favour because they are based upon an old and erroneous notion, that "the cortical centres act only through the corpora striata." How far this is from being a correct representation of my position the reader may judge for himself, seeing that this is the first occasion on which any mention of these basal ganglia has been made in this paper. My views are, in fact, wholly independent

[1] This latter conclusion is based upon deductions made from his observations upon animals. See note on p. 9 of this paper.

[2] *Loc. cit.*, 2nd ed., p. 344.

[3] *Loc. cit.*, p. 381.

of any fixed notions concerning the functions of the corpora striata, as to which so much doubt has arisen of late years.[1]

Facts which favour or oppose the Notion, that the Centres in the Rolandic and Marginal Areas are real Motor Centres.

Having adduced the reasons which induce me to believe, that the so-called "motor centres" of the cortex are in reality the cortical termini of muscular sense impressions, and having discussed, and I hope shown, the futility of some of the objections which have been made to this view, I will now briefly glance (*a*) at the facts upon which reliance is placed by others in support of the notion, that these centres are really motor, and then turn (*b*) to an enumeration of the strong reasons which, in my opinion, can be brought forward against any such hypothesis.

(*a*) The first facts to be considered are the results of stimulation or destruction of the regions in question.

In regard to these it will be well, first of all, to quote what Prof. Ferrier says. He writes as follows:[2]—"As regards the physiological significance of these regions, we have seen that we cannot conclude, from the mere occurrence of movement on the electrical stimulation, that the regions are truly motor; for the stimulation of a sensory centre may give rise to reflex or associated movements. . . . Whether the centres now under consideration are directly motor, or only give rise to movements in a similar reflex, or indirect

[1] This criticism comes, however, all the more strangely from Dr. Ferrier, seeing that in my work 'The Brain as an Organ of Mind,' 1880, I certainly could not have said anything more definite about the functions of the corpora striata than he said in the first edition of his 'Functions of the Brain,' 1876, where I find the following statements:—"The cortical motor centres which necessarily act downwards through the corpus striatum" (p. 210); "The corpus striatum is the centre in which movements primarily dependent on volition proper tend to become organised" (p. 214); "In these cases, and in the dog deprived of its cortical centres, the path from impression to action is not, as in the ordinary course of volition, through the cortical motor centres to the corpus striatum, and thence downwards," etc. (p. 215). These were the statements concerning the corpus striatum which were backed by Dr. Ferrier's authority till about two months ago, when the second edition of his work appeared.

[2] *Loc. cit.*, 2nd ed., p. 347.

manner when stimulated, is a question which has been answered differently by different physiologists. The definite purposive character clearly perceivable in most of the movements, however, their correspondence with the ordinary volitional activities and individual peculiarities of the animals, and above all their uniformity and predicableness, harmonize best with the hypothesis, that they are the signs of the artificial excitation of the functional activity of centres immediately concerned in effecting volitional movements, *and as such truly motor.* If these centres are part of the mechanism of volitional movements, then paralysis of voluntary motion, *and of motion only,* ought to result from their destruction, and any apparent exception must be capable of satisfactory explanation in accordance with this view, if it is the correct one." This is in my opinion a thoroughly just statement in all respects, except in regard to the two passages which I have caused to be printed in italics.

To the first passage so printed I altogether demur, as being a complete *non sequitur.* The movements excited might also be expected to have all the characters described by Dr. Ferrier, if they were sensory centres "immediately concerned in effecting volitional movements," viz., those in which "muscular sense" impressions are registered. It is, in fact, a rule which obtains throughout the nervous system, that all motor centres whatsoever are always stimulated into activity through incitations coming from sensory centres or nuclei, consequently the electrical stimulation of such centres or groups of cells, or of the fibres issuing from them, should always evoke just such movements as the excitation of these centres or cells is accustomed to produce. Similarly, destruction of such centres or nuclei should render impossible all such movements as had previously been evoked by their agency as necessary factors.

Secondly, if "these centres are part of the mechanism of volitional movements," and if they subserve such functions as I have imagined, the second passage which has been underlined would also be altogether wrong; their destruction, in that case, would involve not only paralysis of voluntary motion but also loss of the so-called "muscular sense" in the related parts.

So far, then, there is nothing which may not be explained by my hypothesis with just as much ease as by the more fashionable notion. We come, however, now to another set of facts, which are pointed to triumphantly by Ferrier and his adherents, as a crowning proof of the truth of their notions, viz. to the fact, that the study of descending degenerations in the pyramidal tract conclusively shows, that efferent fibres pass continuously from the centres in the Rolandic and marginal areas downwards to motor centres in the medulla and throughout the whole length of the spinal cord.

To this I reply, that the direction of secondary degenerations affords good evidence as to the efferent or afferent functions of the nerve fibres which are so affected, and that our present knowledge shows conclusively that the fibres in the pyramidal tract are efferent fibres—a notion which I hold equally with Dr. Ferrier. This, however, does not at all touch the question, whether the ganglion cells which exercise a trophic influence upon such efferent fibres form constituent parts of sensory or of motor centres, which is the real question in dispute. If this trophic influence is a mere collateral incident of the functional activity of the cell, as is now generally supposed,[1] then it must always be exercised in the direction taken by currents starting from the cell, so that the fact, that secondary degenerations in the pyramidal tract pursue a descending course, merely tells us that we have to do with efferent nerves, and absolutely nothing as to whether the trophic cells from which they issue belong to sensory or to motor centres.

The direction of the degeneration says nothing, therefore, which is more in favour of Ferrier's than of my hypothesis.

Again, the fact that these pyramidal fibres pass directly downwards from the centres in question in the cerebral cortex to groups of motor cells situated in the pons, the medulla, and throughout the spinal cord, is a point which tells no more in favour of the one hypothesis than of the other.

Thus, it is an essential part of my general view (which I stated so long ago as 1869) to suppose, that the elementary motor mechanisms which are called into play in voluntary

[1] And as Dr. Ferrier himself imagines (*loc. cit.*, 2nd edit., p. 85).

movements are the same, and no other, than those which have to be called into action in corresponding automatic movements (see p. 65). This view has lately been strengthened by some valuable experimental researches of Ferrier and Yeo, concerning which the former says[1]:—"The stimulation of each motor root of the nerve plexuses of the limbs in monkeys, calls forth combined movements involving the co-operation of numerous muscles, widely separated from each other anatomically, but all resulting in actions such as are seen to be constantly associated together in the ordinary modes of activity of the animal." After giving illustrations of this, Ferrier adds:— "These facts render it probable that each segment of the cervical and lumbar enlargement of the spinal cord, whence nerves proceed to the limbs, is a centre of co-ordinated synergic muscular movements, of a character adapted to the habits and requirements of the animal in its ordinary modes of activity." Ferrier's recent work, therefore, gives a positive basis of support to the view which I had previously enunciated, and have lately reiterated,[2] though it seems to me to tell rather against his notion that there are separate motor centres in the cortex, on the ground that the spinal mechanisms merely require excitations from the cerebral cortex in order to evoke purposive movements.

Either view as to the functions of the cortical centres would still, and equally, leave in doubt the question, whether or not other centres in any way co-operated with these and the medullary or spinal centres in the bringing about of new and complex voluntary acts. Of course we know that, where station and locomotion are concerned, the cerebral incitation must be in part devoted to a rousing of activity in the conjoined mesencephalic and cerebellar centres. Again, supposing the thalamus and corpus striatum to constitute a higher couple still for the performance of acts which are not voluntary, as Dr. Ferrier suggests, we may ask what are the efferent fibres through which these centres act upon those below, and we may put the same question in regard to the combined mesencephalic and cerebellar centres. Such questions become all the more

[1] *Loc. cit.*, p. 76.

[2] 'The Brain as an Organ of Mind.' 1880, p. 558.

pertinent if applied to the case of a dog which has had its so-called "motor centres" removed from both hemispheres, and which, if it should live long enough, as in Carville and Duret's experiments, would presumably have secondary degenerations throughout both pyramidal tracks. Yet in such animals, after a time, both station and locomotion become possible.[1] Here, therefore, the incitation of all the motor nuclei needful for the accomplishment of the complex muscular contractions occurring during locomotion must take place through channels other than those pertaining to the degenerated pyramidal tracts. I make these latter remarks merely for the purpose of indicating how much we still have to learn upon this whole set of questions, and not with the view of showing that such difficulties press more heavily upon the one than upon the other of the two hypotheses now being weighed in the balance.

The above-mentioned are the only real grounds that have ever been put forward in support of the notion, that the centres in question in the Rolandic and marginal areas of the cortex are true "motor centres."[2] What has been said above, however, clearly shows that the facts admit of a totally different interpretation. If we put aside for the moment the question, whether the "muscular sense" is or is not impaired or lost in limbs, less or more paralysed from lesions occurring in these portions of the cerebral cortex, all the remaining facts are, to say the least, fully as much in accordance with my view as with that of Dr. Ferrier. If, however, subsequent observations on man should confirm what the observations of Horsley and Zenner render possible or even probable, that loss or impairment of the "muscular sense" will be found to exist in the paralysed parts in cases of cortical disease, then the balance of evidence would be completely turned in favour of my hypothesis, that these Rolandic and marginal centres are the cortical termini for "muscular sense" impressions. This would be crucial evidence which could not be gainsaid even by the

[1] Ferrier, *loc. cit.*, p. 367.

[2] The mere large size and other peculiarities of the nerve cells in these regions of the cortex may be explained by the one as well as by the other hypothesis, if we look to the nature of the stimuli issuing from them, and to the length of the paths which such stimuli would have to traverse.

most sceptical, since as Dr. Ferrier himself puts it, in accordance with his view that the above-mentioned cortical centres are true motor centres, "paralysis of voluntary motion, and *of motion only*, ought to result from their destruction."

(*b*) So far, however, I have only been discussing the value of the evidence which its supporters put forward in favour of their notion, that the cortical centres above indicated are true motor centres; it still remains for me to state, what can and ought to be said against the notion of the existence of any such centres in the cerebral cortex. Whilst, in fact, the additional evidence from observations on man, alluded to above, may be needed to convince the strongest partizans of the "motor" hypothesis as to the truth of my particular view, I trust many persons may be found to agree with me that, even in our present state of knowledge, the following considerations are amply sufficient to show, not only that there is now no independent foundation for the hypothesis that "motor centres for voluntary action" exist in the cerebral cortex, but that such a hypothesis is in reality repugnant alike to physiological and to psychological data.

The theory which first led to the postulation of the existence of such centres by Dr. Hughlings-Jackson has been thoroughly disproved, and nobody admits this more fully than does Dr. Ferrier himself. Hughlings-Jackson has always strongly supported the view of Bain as to our feelings of movement being "concomitants of the outgoing current," and that such feelings are ideally revivable in motor rather than in sensory centres. The logical corollary of these views is, undoubtedly, that motor centres should exist in the cerebral cortex. As we have seen, Dr. Ferrier has, one by one, dropt these fundamental doctrines as erroneous, and yet he still clings to a theory which is the natural associate of such rejected doctrines. His position is a very inconsistent one.

The supposition that motor centres exist in the cortex for the performance of voluntary movements, is, however, thoroughly repugnant in itself to what we may term the physiologico-psychological analysis of the volitional act. This teaches us that sensory centres are the real guides of volitional action; that their activity corresponds with the very essence of volition;

that they, in fact, do just such work as that which those who do not adequately think out the problems involved attribute to cortical motor centres.

Ferrier's view is equally repugnant to the physiology of movements from an evolutional standpoint, which teaches us that the self-same motor mechanisms which are at first called into play volitionally (that is, by incitations from cortical sensory centres), become, with lapse of time in new generations of animals, the motor mechanisms for corresponding automatic acts. Ferrier's view, that the centres in the cortex are motor, expressly says that they are for the accomplishment of volitional acts only; and at the same time he tells us nothing as to how this assumed registration of voluntary movements, in regions altogether distinct from those in which automatic movements are registered, can be reconciled with the fact, that every sort of transition exists between voluntary and automatic movements, and that many of the self-same movements are at times executed volitionally, though at other times they are performed as typical automatic movements. If he does not mean to say that the voluntary acts are registered in the cortical centres as actual motor mechanisms, then I maintain that, in consequence of an imperfect analysis of the phenomena of volition, he is simply attributing to assumed motor centres that guiding action which is really carried on in sensory cortical centres.

Finally, there is only one complete set of excitable areas in the cortex of each hemisphere (through which movements may be evoked in all parts of the body), but if Dr. Ferrier's views were true there ought to be two complete sets of such excitable centres in each hemisphere. Clearly and indubitably the sensory incitations to movement, constituting (as all admit) part of the volitional act, *must pass off from certain cortical areas in a definite and orderly manner in order to excite motor centres, wherever they may be situated.* I assume that they pass off in such fashion from the cortical termini for "muscular sense" impressions. The stimulation of these centres or efferent fibres, which must exist, should, therefore, clearly be capable of evoking purposive volitional movements. All those who, contrary alike to the teachings of psychology and of

physiology, would look for separate volitional motor centres in the cerebral cortex must seek to discover therein another distinct set of excitable areas. Perhaps it might be easier and more conducive to sound doctrine for them to reconsider the whole question?

It may be supposed by some to be, after all, a matter of no importance, whether we call these areas in the Rolandic and marginal regions of the cerebral cortex "motor centres" or not, especially seeing that, as I myself admit, they are the areas whence volitional motor incitations issue, so that their efferent fibres really convey motor incitations.

To this position I altogether demur. It is, in fact, a matter of the highest physiological and psychological importance that the correct view should be recognised and that a nomenclature which was, as I am disposed to think, entirely based upon an incorrect and mistaken view should be altogether discarded. There should be no half measures; the old view is either right or wrong, and if wrong it cannot be right to designate certain sensory areas of the cortex either as "motor" or "psycho-motor" centres, even though they are the cortical termini of ingoing impressions resulting from movements. I would put it therefore in this way:—

1. For the sake of physiological consistency, we should not call a cortical centre for afferent impressions "motor," *any more than we should call the cell nuclei on the sensory side of a spinal reflex arc "motor."* In each case they give birth to fibres which convey motor impulses, and in each case the stimulation of such internuncial fibres would give birth to definite movements.[1]

2. Again, the retention of any nomenclature which implies that the excitable areas in the cerebral cortex are "motor centres," tends to foster false physiological and psychological doctrines such as these:—

[1] As I have elsewhere said ('The Brain as an Organ of Mind,' 1880, p. 585):— "The plan on which nerve centres generally are constructed, of whatsoever grade, makes it essential that the stimulus which awakens the activity of a 'motor' ganglion or centre shall come to it through connecting fibres from a 'sensory' ganglion, centre, or knot of cells—that is, from cells which stand in immediate relation with ingoing fibres." The connecting fibres which I term "internuncial" (*loc. cit.*, p. 586), Schiff speaks of as "kinesodic."

(*a*) The notion that we have in the sense of movement to do with a so-called "active sense," differing altogether in kind from other modes of sensibility ("passive senses"), seeing that its impressions are wrongly imagined to be "concomitants of the outgoing current."

(*b*) The notion that "mental operations in the last analysis must be merely the subjective side of sensory and motor substrata";[1] that we have such things as "motor ideas"; that "movement and sensation are the stuff of which our mental life is composed"; or that "at the root of our mental life, everywhere and always, there are movements."[2] If the activities of real motor centres are simple physiological processes (see p. 48) devoid in themselves of all psychical accompaniments, then the first phrase is an altogether erroneous one; while the subsequent phrases or statements are equally misleading, since they all imply that motor centres have such psychical accompaniments, and moreover confound them with the only real ideas of movement which we possess, viz., revived kinæsthetic impressions. As the writer has elsewhere said[3]:—"If the various impressions which go to make up the kinæsthetic sense are all of them (as we suppose) real 'ingoing' impressions that traverse different kinds of sensory nerves, the mere difference of the mode or occasion on which they are excited should not lead to them being spoken of as though they were radically different in nature from other sensory impressions. So that in accordance with this view, the dictum '*nihil est in intellectu, quod non fuerit prius in sensu*,' loses none of its old force."

It must not be supposed, moreover, that the evil consequences of the above-mentioned erroneous notions are limited to the spheres either of physiology or psychology. They are far-reaching in their effects. The associated doctrine, that words are revived in thought as "motor processes," tends, in my opinion, to throw confusion into the sphere of practical medicine by hampering the proper comprehension of the

[1] Hughlings-Jackson, in 'Clinical and Physiological Researches on the Nervous System' (reprint), 1876, pp. xx.–xxxvii.

[2] Ribot, in "Les mouvements et leur importance psychologique" ('Revue philosophique,' Dec. 1879).

[3] 'The Brain as an Organ of Mind,' 1880, p. 597.

great class of speech defects, and thereby, as I have attempted to show elsewhere, tends to check the advance of our knowledge of cerebral localisation in that direction.[1]

For all the reasons which have been above set forth, therefore, it seems to me to be a matter of extreme importance to recognize, that the excitable areas in the Rolandic and marginal regions of the cortex are in no proper sense of the term "motor centres," and that the evidence at present in our possession makes it extremely probable that they are termini for kinæsthetic impressions derived from muscles, so that their excitation in this or that region is the immediate precursor of this or that kind of voluntary movement.

DISCUSSION.

Dr. Ferrier.

Dr. Bastian has gone over much ground where it is unnecessary for me to follow him. I am, however, glad to be able to say, that in many points I am thoroughly in agreement with him, and in particular in all that relates to the sense of movement being dependent on centripetal impressions, and not on "outgoing" currents as contended for by Bain, Wundt, and others. I also admit that in the first edition of my work on the 'Functions of the Brain,' I used expressions in reference to the subjective aspect of motor substrata which appeared inconsistent with the views I had elsewhere enunciated on this topic; but I have made no change of front, and am glad that in the second edition of my work I have succeeded in clearing away any misconception that may have existed as to my real meaning, which is, that I consider that the activity of motor centres and motor nerves by themselves is entirely outside the sphere of consciousness. Their activity is revealed in consciousness only through the coincident functioning of sensory nerves and centres. But I must join issue with Dr. Bastian on almost everything else that he has said in reference to the nature and cortical localization of the "so-called" muscular sense. I use the expression "so-called," because I think it is a misleading term. What is merely a complex assemblage of impressions of different categories, has no claim to be regarded as a

[1] See p. 50.

sixth sense, even though we admit in the fullest manner that the sensations connected with movement and muscular strain greatly extend the range of our appreciation of weights and resistances, beyond that founded on mere cutaneous pressure.

I should prefer the term "sense of movement," or even adopt Dr. Bastian's term "kinæsthesis," if I might do so without adopting the meaning he attaches to it. The sense of movement comprises impressions derived from the skin, fasciæ, tendons, joints and muscles themselves, conditioned by the act of movement. But the muscular fibre itself is of little or no moment in this relation; for the sense of movement and degree of strain passively communicated to a limb, may remain, to all intents and purposes, unimpaired, when the muscular fibre, motor nerves, and anterior horns of the spinal cord are entirely degenerated, as may be easily demonstrated in any incurable case of anterior poliomyelitis.

I entirely demur to inclusion in the muscular sense any "unfelt," or, as Dr. Bastian now prefers to say, "little felt" impressions. The sense of movement is an act of conscious discrimination, and I am unable to understand how unfelt impressions can affect the sum of this consciousness. Unfelt impressions are necessary for co-ordination of movements, but co-ordination of movement and sense of movement are two totally distinct things. As a rule, the sense of movement accompanies all movements, but we may have defects of co-ordination without defects in muscular sense, and loss of muscular sense without inco-ordination.

Illustrations of the first of these conditions are furnished plentifully by cases of locomotor ataxy, in which the sense of movement is retained in its integrity notwithstanding the most profound disorders of co-ordination. Of the second condition we have a crucial instance in the well-known case of Remigius Lens, quoted by Dr. Bastian (p. 14). Here we have a case in which, notwithstanding absolute cutaneous anæsthesia and loss of all sense of movement, the individual was able, with the aid of vision, to co-ordinate his movements with perfect precision, and even without the aid of vision, to use his hands with a very fair degree of steadiness. "If he is asked to raise his foot to a given height while his eyes are shut, he accomplishes the act by a perfectly quiet and suitable motion" (p. 15).

Dr. Bastian regards this as an exceptional case. In one sense it is so, but it is to my mind a crucial experiment proving that co-ordination is possible in the entire absence of any sense of movement. That in general the removal of the sensations accompanying muscular action should for a time lead to marked

uncertainty of movement is only what we should expect; but the point of this case is the demonstration, that they are not essential to accurate co-ordination, and may be entirely compensated for by visual sensations and visual ideas of movement. Again, in hemianæsthesia, whether functional or organic, there may be complete abolition of all kinds of sensibility, cutaneous as well as muscular, with complete retention of motor co-ordination. The movements of the hemianæsthetic are not inco-ordinate or ataxic.

I confess I am amazed at Dr. Bastian's statement (p. 19), that in hemianæsthesia "there is generally no very appreciable loss of 'muscular sense.'" I have myself examined several cases of this kind, and I have read of many more, and I do not know a single observer, with the exception of Dr. Bastian, who has ever seen a case of hemianæsthesia in which muscular sense was not abolished, or impaired in proportion to the degree of the anæsthesia existent. If Dr. Bastian is right in regarding mere co-ordination of movement as proof of the existence of muscular sense, then we might admit his conclusion. But his argument, in face of the facts mentioned, is a manifest *petitio principii*. I have elsewhere stated[1] that defects in muscular sense are usually associated with defects in common or tactile sensibility, and that I am not aware of any case in which there has been loss or impairment of muscular sense without coincident impairment of tactile sensibility. That it may not occur I will not deny, but I deny that it has been satisfactorily proved. It is stated by Brown-Séquard, and generally accepted, that there is loss of muscular sense on the limb on the side of hemisection of the spinal cord, and retention of this sense on the other limb which is otherwise entirely devoid of sensibility. I find that the only ground for this assertion is the fact, that the anæsthetic limb is directed with fair precision in its movements. On this fact the hypothesis is based, that the muscular sense is retained, because it is believed that the power of directing movements is dependent on the muscular sense—a conclusion which I hold to be entirely without foundation. The power of directing movements has no necessary connection with the muscular sense, and may be perfectly well retained when there is not a vestige of any sense of movement.[2]

In proof of the retention of muscular sense, in spite of profound impairment of tactile or common sensibility, Dr. Bastian refers to some observations by Leyden and Bernhardt. Leyden[3] records two cases of ataxy, with great loss of tactile sensibility, in

[1] 'The Functions of the Brain,' second edition, p. 64.
[2] See "Hemisection of the Spinal Cord." 'BRAIN,' April 1884.
[3] Virchow's 'Archiv,' Bd. 47.

which nevertheless the power of appreciating differences in weight was not lost. But he makes the very important statement, that these patients could only discriminate between *heavy* weights. And I have elsewhere endeavoured to show,[1] that the power of discrimination in these cases was probably dependent on the degree of general bodily strain, altogether apart from the sensibility of the limbs themselves to which the heavy weights were attached. Bernhardt's statements do not differ from those of Leyden. Such cases lend little support to Dr. Bastian's views. In reference to the next statement, that muscular sense may be lost in presence of normal tactile or common sensibility, I must say that I have myself never seen such a condition. Nor has Leyden. In investigating the tactile sensibility of a limb, I never consider that this is normal unless the slightest touch is clearly perceived and accurately localised. Unless this is distinctly proved and recorded, I refuse to accept any mere assertion, that the tactile sensibility was normal in any case. A prick of a pin may be felt, or a pinch, or pressure, when there is profound impairment of tactile sensibility and localisation of impressions, notwithstanding. And considering the great carelessness which characterises clinical records in the investigation of tactile sensibility, I decline to accept statements as to tactile sensibility being normal, unless we have good reason for believing that it has been satisfactorily investigated.

Yet I find that Dr. Bastian, with all his experience, is able to adduce only two cases, both hysterical, in support of his assertion, that muscular sense may be lost apart from affection of tactile sensibility. In the first of these cases (p. 23) sensibility was said to have been absolutely intact. In the second (p. 24) "sensation of contact and of temperature preserved; sensations to pain obtuse and abolished in places." The second case may therefore be eliminated, and all that remains is the first.

For the reasons above stated, I decline to accept this case without further confirmation. As there was no post-mortem examination in Zenner's case of supposed injury to the Rolandic area alluded to by Dr. Bastian (p. 25), it is useless to argue about the position and extent of the lesion, though it certainly confirms my statement of the intimate relation between cutaneous and muscular sensibility. "Cutaneous sensibility is good everywhere excepting over the fingers. . . . *The knowledge of the position of the fingers is impaired.*" The knowledge of the position of the other parts was presumably normal, notwithstanding their paretic condition.

[1] The 'Functions of the Brain,' second edition, p. 392.

As to the cortical localisation of the so-called muscular sense, I hold that the centre for this and for all forms of tactile and common sensibility is the falciform lobe; and that there is no loss or impairment of tactile or muscular sensibility except in connection with direct or indirect affection of this region, or of the sensory tracts of the internal capsule. The motor centres are distinct from the sensory as much in the cerebral cortex as in the spinal cord. The evidence in favour of the motor character of the Rolandic zone is precisely of the same nature as that in favour of all other centres and tracts termed motor. Excitation causes movement, and destruction causes paralysis of movement, *and of movement only*. Dr. Bastian objects to the evidence on this head drawn from experiments on the lower animals only. I myself have always insisted on the importance of clinical investigation in all matters relating to subjectivity, and I am quite content to take Bastian on clinical ground alone. I have seen cases of cortical lesions of the motor zone—cases of aphasia—in which the individuals have been able to indicate with the left arm every position passively communicated to their paralysed right.

Dr. Bastian has attempted—with, in my opinion, poor success—to extract support of his views from the cases of operation for cerebral tumour, which we have recently had in Queen Square. In the case of James B. (p. 27), who was under my care, there was no affection of sensation, though a decided degree of right hemiplegia, before the operation. The operation was of such a nature as to involve the gyrus fornicatus or its tracts. Hence the slight amount of impairment of tactile sensibility, *and with it of the sense of position of the fingers.*

In Dr. Jackson's case of Thomas W. (p. 29) there was still clearer evidence of injury of the sensory tracts. For after the removal of the tumour from the right parietal region, which resulted in paralysis of the left arm and slight paralysis of the face, there was *tactile anæsthesia over the whole of the left side of the body.* Unless we are to suppose, therefore, that the motor centre of the arm is not merely the "kinæsthetic" centre of the arm, but also the tactile centre of the whole of that side of the body, we may say that beyond all doubt in this case the sensory tracts of the internal capsule were injured, which they might readily be from the nature of the disease, and the deep incision necessary to remove it.

But what will Dr. Bastian say to the following case, at present under my care in the hospital? It is the case of J. H., who in January of this year began to suffer from left-sided convulsions, followed by hemiplegia, dependent on cerebral tumour affecting the Rolandic zone of the right hemisphere. At the time of ad-

mission into the hospital on Sept. 22, he was usually in a semi-comatose condition, but occasionally he woke up and was able to answer questions. From the notes of his case in the clinical record I extract the following:—

"*Movement.*—Cannot move the left arm or leg at all. Face-paralysis almost complete, at lower part on left side. Angle of mouth moves a little when he tries to show his teeth. Can close and screw up his left eye well. Tongue protruded well, but deviates to left side.

"*Sensation.*—Normal. Patient feels a touch with a quill readily over both sides, and can describe where the stimulus is applied. Painful sensation also normal. Was able to localise sensation readily and accurately. Muscular sense also normal."

On September 23rd, Mr. Horsley, at my request, removed the tumour. This had a diameter of nearly three inches, and occupied the middle of the Rolandic zone and invaded the cortex, and subjacent medullary fibres, to a considerable depth.

On September 24th, the clinical record reports as follows:

"Patient quiet, perfectly conscious. . . .

"Left leg and arm remain completely paralysed; face is also paralysed, but not quite completely. Sensation practically normal over whole of left side; though sometimes patient refers a touch to the nearest joint above the part stimulated, *e.g.* when the hand is touched, he refers to the wrist; when the forearm, to the elbow. This is not always so, as sometimes he points to or localises quite correctly the part touched. Muscular sense good; he can describe fairly well the position of his limbs.

"*Sept. 25th.*—Condition of sensation and reflexes the same as yesterday.

"*Sept. 26th.*—Condition much as yesterday.

"*Sept. 27th.*—Sensation slightly defective on face and arm or left side, and perhaps over leg, *i.e.* that patient generally, but not always, feels a light touch with the feather of a quill, and can often localise it correctly; but sometimes refers it to some other spot near, as when touched on the back of a finger, he thought it was the back of his hand. Muscular sense normal.

"*Sept. 28th.*—Always feels a touch, but does not localise it correctly. Touches on all fingers of left hand he refers to the thumb; on the back of the hand to wrist. But his answers vary, and sometimes he appears to localise correctly. On face and leg the localisation is usually accurate. Muscular sense perfect as to position, but when a finger is bent, although he says it is so, he often does not recognize which finger it is.

"*Sept. 29th.*—Refers touch on fingers to the thumb, as he did

yesterday. On face, sensation not quite as accurate as on right side. Sometimes he does not perceive a very light touch with the feather of a quill. Over leg and foot sensation is normal. Muscular sense normal."

After this date the patient began to experience some pain and tension in the region of the wound, and a considerable swelling ensued.

On October 6th sensation, cutaneous and muscular, was found to be considerably impaired over the whole of the left side.

The patient recovered so as to be able to leave the hospital; hemiplegic on the left side, totally as regards the arm, but partially only as regards the leg. Some degree of anæsthesia to light contact, and impaired localisation specially on arm and hand remained.

The facts of this case are of the utmost importance in reference to the question before us. Here we have a case demonstrably of cortical and subcortical disease in which, with the most complete paralysis of motion, sensation, cutaneous and muscular, was perfectly normal. Even after removal of the disease, at a time when any positive disturbances of sensation might readily have been ascribed to general perturbation of the brain, and not to the local lesion, we have for several days practically perfect retention of cutaneous and muscular sense.

It is true that, later on, coincident with certain morbid changes which showed themselves in the wound, and which I at the time ascribed to further extension (since verified) of the original lesion, a considerable degree of anæsthesia manifested itself on the left side. But this might well be from implication of the sensory tracts of the internal capsule; and the fact remains, that an enormous amount of the motor area of the right hemisphere was destroyed without in any appreciable degree impairing the sense of movement of the limbs entirely paralysed as to motion. This I hold to be a crucial experiment, which absolutely knocks the ground from under Dr. Bastian's feet.

The facts which I have brought forward prove, that the motor centres are motor centres in precisely the same sense as other motor centres. Excitation causes movement, destruction causes paralysis of movement, and of this only. Further, when these centres are destroyed, degeneration proceeds in the pyramidal tracts, precisely as it does in the motor nerves when the anterior horns are destroyed. If Dr. Bastian's theory of these centres is correct—that they are only excito-motor of true motor centres in the corpus striatum or spinal cord—then the pyramidal tracts must be merely afferent or intracentral. But inasmuch as he

expressly admits that they are efferent and convey motor impulses, he appears to me to admit all that is characteristic of the activity of motor centres. Just as the activity of the anterior horns of the severed spinal cord is displayed in response to stimuli proceeding from the posterior roots and horns, so the motor centres of the brain are thrown into activity by stimuli proceeding from the sensory centres—visual, auditory, tactile, &c. As the motor nerves convey stimuli to the contractile muscular fibres, so the pyramidal tracts convey impulses to the motor apparatus—not perhaps directly to the muscular fibre—but to the neuro-muscular mechanism by which actions are capable of being effected. As I have elsewhere ('The Functions of the Brain') discussed at some length the mechanism of volitional movements, the basis of our ideas of movement, and the relations of the motor centres to these and to motor acquisitions, I do not consider it necessary to discuss these questions further on the present occasion.

Mr. Sully.

Mr. Sully remarked that though he felt the force of the facts and arguments in favour of the view, that the muscular sense was, on its physiological side, essentially afferent, he could not accept this as a complete theory of the subject. Among other facts which seemed to conflict with this view were the hallucinations that were known to follow the loss of a limb. When in these circumstances a patient was able to give a certain degree of definiteness to his feeling of the (imagined) situation, and movements of the missing member, this appeared to point to the co-operation of the efferent nerve-process as a factor in the muscular sense. It is not unlikely that the so-called muscular sensations are highly complex psychical phenomena, in which afferent currents play a prominent part in giving the exact measure of direction, rapidity, and distance of movement, though an essential, and perhaps the most characteristic element, is supplied by the efferent process of innervation itself.

Dr. Ross.

Before entering on the general discussion, I shall endeavour in a few words to explain the difficulty started by Mr. Sully, so as to bring it into accord with the theory, that the sense of muscular movement is correlated with the activity of in-going and not of out-going currents. When a person suffering from a profound degree of hemianæsthesia, say of the left half of the

body, is asked to move his arm, he makes an effort to comply with the request. He *knows* that he has made the effort, and *believes* that this effort has been effective in moving the left arm in a particular direction. If, however, the patient's eyes be closed he has no means of verifying his belief, but he still knows that he has made the effort. But, as has been first pointed out by Dr. Ferrier, the sense of effort consists chiefly of the feelings which accompany that closure of the glottis and arrest of respiration which is necessary to give fixity to the muscles which find their origin in the bones of the trunk. Now the muscles of respiration are bilaterally associated, and the activity of the one hemisphere—the right in the supposed case—sets in motion the muscles of both sides, and the sense of having made an effort would be awakened by currents passing inwards along the centripetal nerves of the right or healthy respiratory muscles and of the right side of the glottis. That this theory affords a better explanation of the facts than the one which seems to be favoured by Mr. Sully, viz. that the sense of effort is correlated with the out-going stream, is much strengthened by the case of hysterical anæsthesia quoted by Dr. Bastian on page 16, from Duchenne and Briquet. "If," say the authors, "after having made her squeeze the hand, one prevents her seeing it, and tells her to cease all effort, one feels that the contraction of the flexors continues, and it is necessary to employ considerable force to open her hand." It will be generally admitted that the persistent contraction of the flexors in this experiment was caused by a continuous outflow of energy from the cortex, and yet the patient remained quite unconscious of making any kind of muscular effort.

Most of the questions under discussion to-night are questions of interpretation and not of fact, but there is one issue between Dr. Bastian and Dr. Ferrier which is one of fact and not of interpretation. Dr. Bastian says that in hysterical hemianæsthesia the muscular sense is almost always preserved, while Dr. Ferrier asserts strongly that it is lost. I must myself plead guilty to having stated, in more than one publication, that in such cases the muscular sense is lost, but I have in recent years observed many cases in which it was preserved. A few years ago I had a patient under me who had had an attack of left-sided hemiplegia, in which the leg was more paralysed than the arm, and as usual in such cases the motor paralysis was accompanied by hemianæsthesia. In this case it may be presumed that the symptoms were caused by a lesion, probably a hæmorrhagic focus in the lenticular nucleus and implicating the posterior part of the internal capsule. When a book was placed in this patient's

hand she held it quite well so long as her eyes were fixed upon it, but on her attention being diverted by getting her to put out her tongue, the grasp immediately relaxed and the book fell, but the patient continued to hold out her hand horizontally as if she were still supporting the book. In her case the muscular sense was lost. I have tried this simple experiment in many cases of hysterical hemianæsthesia since this patient was under observation, but have not met with one who dropped a book or other weight from the anæsthetic hand on the eyes being closed. It seems to me, therefore, that in most cases of hysterical hemianæsthesia the muscular sense is retained, although I have no doubt that it may be lost in aggravated cases. But if the muscular sense is lost in any case of hemianæsthesia without this loss being accompanied by a corresponding degree of motor paralysis, the argument which Dr. Ferrier has founded upon the fact is as valid as if this sense were lost in all cases of the kind. In carrying on a discussion upon any controverted subject, the upholders of a certain theory ought to adduce arguments the validity of which will be acknowledged by their opponents, and it must be acknowledged that although Dr. Ferrier's argument carries conviction to my mind and to the minds of those who accept his theory of psycho-motor action, yet it is not likely to be equally convincing to those who reject his theory. It is still open to Dr. Bastian to reply, and indeed he does reply, that although it be granted that the muscular sense is sometimes lost in the absence of motor paralysis, yet this loss is caused by injury of the centripetal conducting paths and not by destruction of the kinæsthetic centre itself. When the centre itself is destroyed, it causes, according to Dr. Bastian's theory, an equal degree of motor paralysis and of loss of the muscular sense. I repeat that I do not agree with Dr. Bastian, but I fear no evidence which I can adduce in reference to this point will suffice to drive Dr. Bastian from the position which he has occupied, and compel him to agree with our views.

It is a trite remark, at least since the days of Locke, that most of men's disputations arise from the use of ambiguous terms, and certainly the terminology adopted by Dr. Bastian seems to me to favour every species of fallacy and confusion. Take, for example, his nomenclature of animal movements, which he divides into five classes, viz. (1) reflex or automatic, (2) secondary-automatic, (3) instinctive, (4) ideo-motor, and (5) volitional. In this classification, reflex and automatic are used as synonyms for the same kind of movement. Here, then, there is a merging together of two valuable terms, one of which is well adapted to express the kind of movement indicated, while the other ought to be reserved

for some other kind of movement. It is possible that no great confusion might result if these words were always consistently used as synonyms for the same kind of action, but they are not. The element of confusion arises chiefly through the use of the term *secondary-automatic*. Dr. Bastian argues that voluntary movements tend by frequent repetition to become first ideo-motor and then secondary-automatic, but whether or not he makes any distinction even of degree between the movements indicated by the two last terms is not very clear. Now if a voluntary movement may by frequent repetition become secondary automatic, and if automatic and reflex are merely synonymous terms for the same kind of movement, it is clear that, in his opinion, a voluntary movement may by frequent repetition, become reflex. This view I should most strenuously oppose. I do not believe that any amount of repetition tends to convert a movement which was once voluntary into a reflex action either in the individual or the race. But the confusion which arises from the improper use of such terms as reflex, automatic, and secondary-automatic is small and unimportant as compared with that which must follow the indiscriminate use of such terms as instinctive motor, ideo-motor, volitional motor, and other compounds of metaphysical and physical ideas of the same class. Surely the psychology of the present day has outgrown the employment of such unscientific terms as these.

It seems to me that the term "impressive" is also used by Dr. Bastian in a very unguarded manner. He employs it at times to indicate the physical changes set up in the peripheral ends of certain nerves by muscular contractions, and at another time as a name for the sensations which accompany the movements caused by muscular contraction. This is by no means an unimportant objection. It is by a similar ambiguous use of the word "sensibility" that the late Mr. G. H. Lewes came to the conclusion, that the spinal cord possesses consciousness. The term sensibility was used at one time by Mr. Lewes as a generic name for the elementary feelings of the mind, and at another time as synonymous with nerve irritability. He found no difficulty in proving that the spinal cord is possessed of sensibility in the sense of nerve irritability, and from this he inferred that it possesses sensibility in the sense of feeling, from which the conclusion inevitably followed that the spinal cord is endowed with consciousness. The example of Mr. Lewes shows that, in passing from the molecular movement of nerve centres to the phenomena of the mind, it behoves psychologists to tread with cautious steps, but I have no hesitation in saying that Dr. Bastian's tread in this domain is not characterised by that care and caution which his eminence as

a writer and thinker on psychological subjects might lead us to expect.

Instead of the complex classification of animal movements adopted by Dr. Bastian, I should propose to divide them into reflex, psychical, and automatic, this division to be made simply from the character of the movements themselves, and without reference to the nature of the neuro-muscular mechanisms by which the movements are regulated and produced. A *reflex* action is an adjustment of the body to a *present* impression. If the palm of the hand of a person in profound sleep be touched by a feather, the fingers close over the feather, and if the sole of the foot is so touched it is drawn away; but in both examples the adjustment is to a present impression. A *psychical* action is one in which a present impression calls forth an adjustment of the body to an anticipated impression. If the word anticipated be objected to as implying consciousness which is not an accompaniment of every psychical action, it may be said that the present impression evokes an adjustment to a future impression. Of psychical actions there are two chief varieties: the one is general, or frequently repeated in the experience of the race and individual, and it is effected in the absence of consciousness; the other is special, or rarely repeated in the experience of the race and the individual, and is accompanied by the highest degree of consciousness called attention. Between the most general and the most special psychical actions there is every degree of generality, and these different degrees are accompanied by corresponding degrees of consciousness. It also follows that actions which are special and attended by a high degree of consciousness in certain races and in children become, by frequent repetition, general in other races and in adults, and are then attended by little or no consciousness. As an example of a psychical action I may adduce my own action in stretching out my hand to grasp that book on the table. A patch of colour reflected from the book falls on the back of my eye—that is the present impression. I stretch out my hand, not to reach the patch of colour or the present impression, but fully expecting that I shall experience certain tactual feelings, and feelings of resistance and weight—these being the anticipated or future impressions. An *automatic* action is exceedingly difficult to define. Taking the literal meaning of the word, it ought to imply an action which is self-evolved or spontaneous. Some physiologists believe that the respiratory movements are regulated by a centre in the medulla oblongata, which by its own spontaneous activity sends out rhythmical impulses to the muscles without itself being influenced by impulses received through centripetal nerves. Did such an action

as this exist it would be justly entitled to be called automatic, but the balance of evidence seems now to turn in favour of the opinion that the respiratory movements are regulated by a reflex mechanism, and physiologists doubt whether an automatic movement in the narrow sense just indicated anywhere exists in the body. The term automatic is often used to designate a self-adjusting mechanism, but every action of the body results from such a mechanism. It must, however, be admitted that the actions which I have named special psychical actions are regulated by nervous mechanisms which are only imperfectly organised, while the general psychical actions are regulated by fully organised nervous mechanisms, and the term automatic is frequently applied, and not altogether unjustly, to the latter of these two. At the same time I much prefer to dispense with the word automatic in this connection, and to use the word general or unconscious psychical actions in contradistinction to the special or conscious psychical actions, and I should reserve the term automatic to another kind of action altogether. The delicate adjustment by which a certain attitude of the body is maintained in changing circumstances might very appropriately be named an automatic action. The maintenance of the erect attitude on board of a ship tossed by the waves is a good example of the kind of action under consideration. The delicate bodily adjustments which are necessary to maintain this attitude cannot be regarded as being either reflex or psychical in character, and consequently the first of these adjustments ought to be differentiated from the last two by having a distinct name assigned to it, and the word automatic is well adapted for the purpose. In any case I object in the most emphatic manner to the word automatic being used now as synonymous with reflex, and again as indicating an unconscious psychical action. Used in this loose manner it is hardly possible to imagine a word more calculated to introduce confusion into the intricate question which is before us for discussion to-night. Dr. Bastian may legitimately reject my suggested use of these words, and it is open for him to define his terms in his own way, if only he is afterwards consistent with his own definitions; but it is not open to him to use words in several senses without making any attempt at a strict limitation of them. If all of us here to-night would but come to an agreement with regard to the meaning of our terms it appears to me that the differences in our opinions would be found to be exceedingly slender, and such as might be put into the proverbial nutshell.

Every one will admit that the nervous mechanisms which regulate psychical actions must possess centrifugal as well as

centripetal conducting paths, and centres which are in relation with the former as well as centres in relation with the latter. The main question which divides us, or at any rate which divides Dr. Bastian from Dr. Ferrier, is the point at which the centripetal conducting paths and centres cease and the centrifugal centres and paths begin. Most of us will at once say that this point is to be found in the large caudate pyramidal cells of the third layer up the cortex. Now if Dr. Bastian says that he will not accept this view it may be difficult to find an argument that will convince him. I will, however, address to him one argument which has great influence with myself, in the hope that it may have some effect upon him also. Dr. Bastian will admit that the molecular activity of the sensory centres is always correlated with feeling or states of consciousness, and he may possibly acknowledge that consciousness is correlated with the activity of cells and fibres which are not yet fully organised, or, as Dr. Hughlings-Jackson has expressed it, with the activity of centres which are "now organising." But the large pyramidal cells of the cortex, with their numerous connections and axis-cylinder process, are already fully organised, and I cannot imagine that the activity of these cells can be correlated in any degree with consciousness, and if not, these cells cannot be regarded as forming part of sensory centres. Whether this argument will or will not have any weight with Dr. Bastian I cannot tell, but I myself attach so much importance to it that were I compelled to abandon this position I should feel obliged to give up almost every idea I have formed of nervous action, and would have to begin the work of reconstruction over again. I think this argument ought to have considerable weight with our President, and were he to give it full consideration I believe it would force him to give up the theory, that the consciousness of muscular effort is correlated with the stream of out-going energy.

But although the presence of large pyramidal cells gives character to the parieto-frontal area of the cortex, yet it must be remembered that in the outer layers of the cortex in this area the cells are small and are either destitute of processes or the processes are only indefinitely connected with one another; in other words the cells are but partially organised, and consequently the activity of these cells is likely to be correlated with some form of feeling. It is therefore possible, I might even say probable, that the cortex of the parieto-frontal area possesses sensory as well as motor functions, and although we find it extremely convenient to speak of this area as the motor part of the cortex, yet this form of expression is more practically useful than theoretically just. I

feel myself wholly unable to pass any opinion upon the question, whether or not the cortical centres of muscular sense are more likely to be situated along with the tactile centres in the falciform lobule or in the superficial layers of the so-called motor area. I at least give my unhesitating adhesion to Dr. Bastian's opinion, when he says that the kinæsthetic centre is the last centre which must be excited anterior to the movement in any psychical action. The book which lies on the table there reflects certain rays of light on my eyes, but my mind instantly conceives the cause of the colour I perceive to be an object possessing certain dimensions and weight, and if I stretch out my hand to seize the book I do not expect to lay hold of the colour, but of something which will offer a certain resistance to my grasp, and require a certain effort to raise it from the table, and these ideas of resistance and effort are the correlatives of the activity of my kinæsthetic centres. The order of excitation in the psychical act of raising the book has therefore been visual conducting path and centre, kinæsthetic centre, motor centre conducting paths and muscles; while on the action being completed there is a final excitation of the connected kinæsthetic conducting paths and centre which is the correlative of the consciousness of the effort put forth in effecting the movement. Dr. Bastian would say that the kinæsthetic centre and the cortical motor centre are identical, but for the reasons already given I cannot assent to this proposition. I think it, however, quite likely that the kinæsthetic and motor centres coincide, in so far as that the former are situated in the two outer and the latter in the third layer of the cortical cells of the parieto-frontal area of the cortex. My mind is quite open to accept this opinion or the one which seems to be favoured by Dr. Ferrier, viz., that the kinæsthetic centres are situated along with the centres for all forms of cutaneous sensibility in the falciform lobule, and I await further experiments and observations to determine the question

SIR JAMES CRICHTON-BROWNE.

If Wundt's notion and that of Bain as to the sense of effort is to be abandoned, or if that sense is to be reduced from a feeling of the energising of motor centres to a feeling accompanying the reception of centripetal impressions generated by the act of muscular contraction, and especially by contraction of the respiratory muscles, I should be glad to have an explanation of the sense of fatigue, in consonance with the new hypothesis and covering ascertained facts. The sense of fatigue is certainly not

dependent on cutaneous sensibility, and it is something altogether different from the muscular sensibility experienced on galvanisation of the muscles. True, it may be argued, that the sense of fatigue is attributable to impressions made on sensory end-organs in the muscles, by carbonic and sarco-lactic acids and other waste products, which accumulate during exercise, but it is to be noted that it may be felt acutely, as in cases of neurasthenia, when the muscles are well nourished and have been long at rest, and when, consequently, they cannot be overcharged with metabolic *débris.* It has always seemed to me that the sense of fatigue, which is expressed, be it remembered, in an indisposition for mental as well as for bodily exertion, is a modification of the feeling of expended energy, dependent on an exhausted state of the motor centres akin to what we find after an epileptic fit. It has seemed to me that the opposite condition to fatigue, that of muscular restlessness or fidgets, the *besoin d'agir*, that keen appetite for muscular activity, which is revealed by caged wild animals when they pace up and down for hours together, which is seen in the exuberant activity of the young, and which all of us have experienced, more or less, after periods of repose or enforced idleness, is dependent on a surcharged or excitable state of the same centres. Again, of course it might be argued that fidgets are to be ascribed to sensory impressions derived from highly nourished and oxygenated muscles in a state of rest, but again I would point that we have these fidgets, or spontaneous outpourings of ebullient energy, in their most marked and persistent form, in the incipient stages of mania, when the muscles have already been over-used and are wasting rapidly, and so cannot be highly nourished or oxygenated or in a condition to originate a craving for further action which under such circumstances I should be disposed to trace to an erithiomic state of the cerebral motor-centres. The feeling of necessity for muscular exercise, which arises periodically or under special circumstances, seems to have no connexion with any antecedent sensation or emotion, or with any stimulus extraneous to the motor centres themselves. I am aware that other explanation of fatigue and fidgets may be advanced. The late Professor Laycock, for example, used to maintain, that they are due to the direct recognition by certain nerve centres of the varying composition of the blood circulating through them; but the explanation which seems best to cover all the facts and is altogether most feasible, is that which connects them with functional changes in the cerebral motor centres. But even if another explanation—a sensory one—of fatigue and fidgets were clearly established, I should not on that account give up the

sense of effort and motor ideas as opposed to ideas of movement, for I cannot divest myself of the belief that as we have perception at the end of the ingoing currents, we must have apprehension at the beginning of the outgoing currents. Perception is not the "be all and end all," and to relegate our whole thought-material to sensory centres, for that is what it comes to, according to Ferrier's most recent theory—is to degrade a large region of the cerebrum from its high estate, and leave it a mere superfluous intrusion in the brain mass. Professor James of Harvard, who is quoted by Ferrier and Bastian, has analysed a voluntary act or movement into four elements. 1. The preliminary idea of the end. 2. The fiat. 3. The muscular contraction. 4. The end felt as actually accomplished. Now of these four elements only one, the fiat, can, according to Professor James and those who think with him, be in any way connected with or dependent on cerebral motor centres. The preliminary idea is sensory, the contraction is muscular, and the recognition of the effect of the contraction is sensory. But the fiat, if it be further analysed, resolves itself very much into a balance of motives or the balance between two groups of sensory impressions, and all that is left for these large and important motor centres in the brain, with their special structure, is a mere motor impulse. But a motor impulse can be got from a reflex centre in the cord, and it would appear therefore that the large area of the hemispheres corresponding with the motor centres, is—if it has no part to play in ideation—redundant or superfluous, or has at least a very humble office to perform. On principles of physiological economy I cannot accept this. A more minute analysis of a voluntary act or movement than that offered by Professor James is, I think, necessary. We must analyse the preliminary idea and find in that sensory and motor factors, a reproduction of ideal movements as well as of ideal sensations. We must analyse the fiat and discover there not merely a haphazard motor impulse, but a nice adjustment of means to the end to be attained. We must analyse the contraction, and distinguish in it the latent period, shortening relaxation, electrical, chemical, and thermal changes. We must analyse the end felt as accomplished, and recognise in it an appreciation of the centric changes which have succeeded to the voluntary movements, as well as of peripheral changes in joints, skin and muscles. And then, having completed our analysis, we must synthetically recombine all our elements and lift them into the higher medium of the unity of consciousness. If I might be permitted to do so, I would take my friend Professor Ferrier, engaged in the performance of one of his own experiments, as a rough illustration or rather as a working model of a voluntary act.

He stands with the electrodes in his hands, and the anæsthetised monkey with its motor centres, or rather with its Rolandic area exposed, lies on the table before him. With that power of prediction which is the true test alike of science and volition, he says, "I will cause the animal to clench its right fist." That is the preliminary idea, and if we examine the form of words in which it is embodied, we shall be able to satisfy ourselves that it is a complex proposition, involving, like every predicate, sensory and motor ideas—the revival, not only of impressions, but of processes, implying not only observation but the knowledge of power. Well, what is the next step that the Professor takes? Why, he applies the electrodes to the tip of his tongue, to ascertain that the current is neither too strong nor too weak, and he shifts the induction coil according to what experience has taught him to be necessary to secure the end in view, and in doing this he represents dramatically, and on the large scale, a factor in a voluntary act which Professor James has ignored—that nice adjustment of and co-ordination of the means used to the end to be attained, that regulation of the jet—without which all voluntary movements would be ataxic or disproportionate or tentative. In the initiation of a voluntary movement sensory impressions from the part to be moved can play no part. They come a stage too late to supply any guidance in determining the amount or intensity of the outgoing current, and unless feeling accompanying it regulates that, it is difficult to understand how it can ever be anything but experimental in the first instance. After regulating his current, Ferrier applies the adjusted electrodes to a point on the left ascending parietal convolution—that is the fiat—the essence it may be said of the voluntary act, and in executing it he sees and moves, and selects in the latter case performing what must be in itself a complex operation. The fiat given, the right fist is clenched; that is the muscular contraction, and after that were the animal not anæsthetised, the effects of that contraction would be transmitted to the brain for further guidance, or for registration there, along with the effects of the discharge in the centres themselves. At one with Ferrier as I am in regarding the Rolandic area of the brain as made up of true motor and not of kinæsthetic centres, I cannot follow him in depriving these centres of their ideational function.

Sir James concluded by congratulating Dr. Bastian on the grasp, subtlety and lucidity of his paper, which would be heartily acknowledged, he said, by those who differed from him most.

DR. HUGHLINGS-JACKSON.

The question seems to me to be an exceedingly large one, and to come to a great extent to this: Do motor elements enter into the physical bases, or anatomical substrata, of mental states, otherwise consciousness? I have for many years thought that they do. Since it is agreed upon that the lower parts of the nervous system are sensori-motor, I think it *à priori* likely that the higher parts, the physical bases of consciousness, are so too. The unit of constitution of the nervous system throughout is a sensori-motor unit. That would seem to me to agree with the doctrine of Evolution.

We have, as medical men, this problem:—Epilepsy being owing to disease of the cerebral cortex, and insanity being owing to a different kind of disease of the cerebral cortex, what is the constitution of "the organ of mind," the cerebrum, whereby two such different things result from disease of it, having regard of course to the distinction between the physical and the psychical? Moreover, we have the two morbid states in sequence often enough. A man during an epileptic fit, if the primary discharge be strong enough, is stiffened into a rigid mass; after the fit he is temporarily insane. Further we have to show what are the anatomical and physiological conditions of the nervous system correlative with will, memory, reason, and emotion (together states of object consciousness). They are, I submit, activities of sensori-motor nervous arrangements—of nervous arrangements representing impressions and movements of parts of the body. We have also to find the physical differences answering to the differences between faint and vivid states of consciousness (object-consciousness). I submit that, neglecting differences in the sensory sphere, since no one denies the sensory element, the former occur during slight activities limited to the highest motor centres, and the latter during stronger activities of those centres overcoming lower motor centres and gaining the periphery. To my thinking, not only are the convolutions in the Rolandic region motor (middle motor centres) but also the convolutions in front, frontal or præfrontal lobes (highest motor centres). The physical process during ideation differs from that during perception (both being states of object consciousness), in that in the former lower motor centres are not overcome, and in the latter are.

There is no *à priori* reason why states of consciousness should not attend activities of motor as well as activities of sensory cells. It may seem easier to believe that sensations attend activities of sensory elements than that they attend activities of motor

elements. The popular psychologist makes a confusion betwixt active states of sensory elements (physical states) and sensations (psychical states). Starting with what is purely physical, active states of sensory nerves, we get at length, in nervous centres, unconscious sensations (a contradictory expression) and then conscious sensations (a tautological expression). These sensations are then compounded in the highest centres into ideas. After that the popular psychologist inverts the process. Now ideas, sensations, and volitions (psychical states) produce movements (physical states); there is the expression "ideo-motor." In reality, movements result from liberations of energy, and not from states of consciousness.

The common expression "ideas of movements" is very objectionable. I would submit that there is no more an idea of a movement (in the sense either of ideas of states of muscles or of any motor or sensory nervous elements representing any combination of muscles) than that, when I think of, or see a colour (coloured object), there is "an idea of" the retina or of any sensory centres representing it. I submit that we have psychical states attending activities of motor elements of the highest centres. For these psychical states there is no name, except in the case of words; words are psychical states attending activities of motor elements representing certain very special and complex articulatory movements. (Strictly the nervous process is always sensori-motor.) I dwell on the motor element, no one denying the sensory element. I contend that when I make a movement with my hand I have a psychical state just as certainly as I have another when I see that red chair. For if I cut my arm off I have a spectral hand remaining; that spectral hand I can move. This (certainly a psychical state although one uses the term "move") attends, I submit, activities of motor elements of my highest centres. When I cut my hand off I have a ghost of it, but of course that ghost was there when my arm was present. When a man who has lost a hand tries to pick up the reins of his horse with his ghostly hand, that "movement" of it occurs during energising, beginning in the highest motor centres. Dr. Bastian would say that it occurred during activity of kinæsthetic centres. I admit, and am assuming, the activity of sensory elements, but I contend that there is activity of motor elements as well. I submit that the expressions "motor ideas," "ideas of movement," and "muscular sense" (if "muscular sensations" be intended) are illegitimate compounds of physiological and psychological terms.

I have often referred to the well-known displacements of external objects, in cases of ocular paralysis, in proof that psychical states

attend activities of motor elements; but I find that these very cases are claimed by those who repudiate the "out-going current doctrine." I suppose the alterations in size of an after-image during changes of accommodation will be similarly claimed. The following, which I used to think a very strong thing favouring the doctrine that mental states attend activities of motor elements in the highest centres—the alteration in shape of after images—will, I suppose, be claimed too. If we obtain an after-image of a square, and then project it on to an inclined sheet of paper, the spectral square becomes oblong. There is here a change in muscular activity, but it may be said that no mental states attend activities of the motor nervous arrangements effecting it, but only of the sensory elements in activity from changes in the muscles put in action. Let us take an extreme case. A man has progressive muscular atrophy to the degree that he has no muscles in the right arm, and no anterior horns for them. Yet he localises a touch, and for the sake of argument I will grant that he does so as well as ever. I suppose the localisation implies movement, in this case the so-called "ideas of movement." It is true that the patient has no muscles, but the whole of his sensory nervous system is intact, and the whole of his motor nervous system with the exception stated. My submission is that the patient localises the impression because, besides sensory activities, there are also activities of his highest motor centres.

PROFESSOR HORSLEY.

As some cases I have published have been introduced into this discussion, I shall be very glad of the opportunity to say something with respect to the representation of the "muscular sense" in the so-called motor region of the cortex. In the first place, let me say that if I use the terms usually employed, apparently with a twofold meaning, I hope I shall not be misunderstood. Thus if I speak of "sensation" I do not mean by that term, in every sense that I may use it, to connote consciousness. We are very much in want of a term to express better what is generally understood by the word sensation. The changes which go on in nerve endings when they are irritated require some other expression than our common word sensation, which conveys the meaning of the conscious perception of the process which is going on peripherally. If therefore I use the word "sensation," I hope to be understood that in a large majority of cases I shall be referring to a conscious perception of the changes in the nerve endings, but I may also mean the changes which go on in the nerve endings

without the accompanying consciousness. Further, a great deal of stress has been laid on the use of the word motor. What Dr. Ferrier calls "motor," Dr. Bastian, for equally good reasons, calls "efferent"; what Dr. Bastian calls "efferent," Prof. Schiff calls "kinesodic." Now it seems to me that of these the term efferent is the more general in meaning, therefore I shall, as far as possible, avoid speaking of "motor," and I shall apply the term afferent to the impulses which originate the so-called motor sense. There is no doubt as to what is meant by the word afferent. It seems to me that so much stress has been laid upon the meaning of these terms as to introduce no little confusion into this matter. Well, with respect to the first point, viz., the cases of my operations which apparently throw light on this question, personally I desire to lay no great weight on these cases. There are not enough of them yet for us to speak absolutely from the evidence they afford. There is among them one case of cerebral tumour, in which I did, as Dr. Ferrier says, make an incision deeply down into the corona radiata. To my mind the knife did not pass down deeper than one inch. Personally I do not consider that that incision injured the fibres going to the so-called sensory tracts of the cortex, but until the patient dies, I would not like to say exactly what was the extent of that lesion, and therefore, as I stated in the description I gave of my cases in the 'British Medical Journal' for Oct. 10, 1886, I would not rely on their evidence further than to regard them as tending to show that the so-called motor region is the centre of muscular sense. I would put aside that case which Dr. Ferrier has quoted of the man Henderson, for although there certainly seemed to be a delay on the onset of the anæsthesia after the operation, the whole surroundings of the case are so very complicated that even after the death of the patient I should not like to adduce it alone in support of an argument one way or the other. When one has a series of something like twenty-five or fifty cases, then I should like to come forward with some more positive statements. These cases however, to my mind, do tend to show there is possibly some representation of the so-called motor sense in the motor cortex. Lastly, we come to the question of the theoretical possibility, that the muscular sense is represented in the so-called motor cortex. It is not for me to enter to-night into questions of much deeper import, questions of philosophic thought. It would be presumptuous on my part, after what has been said before, by Drs. Bastian, Ferrier, Ross, and Sir J. Crichton-Browne; but as regards the anatomy, *i.e.* the mechanism of the function of which we are now discussing, I should like to say a few words. Dr. Ross has referred to the two main histological divisions of the

cortex, viz. the large ganglionic, the so-called motor cells of the fourth layer, and the smaller cells which exist in vast numbers in the rest of the grey cortex, and he believes that consciousness is correlated with the functions of the smaller cells, and not with the larger ones. I would venture to say that such a belief as that is of course a personal matter, but argument of analogy seems to me to be strongly in favour of this contention. But there is another mode of regarding the anatomical facts just mentioned, and that is the possibility of each being correlated to efferent and afferent functions respectively. Of course, theoretically, we may for many reasons regard the few large corpuscles as essentially efferent, and the small cells as essentially afferent in function. For myself, I am quite prepared to hear it proved some years later that the smaller cells have a distinctly sensory aspect, and that the larger cells have a distinctly motor aspect. Until this anatomical problem has been cleared up, we must leave the question entirely open whether the smaller cells are kinæsthetic or not. Dr. Ferrier would say absolutely that they are not; that there is no representation of sensation in this "motor" region, and that therefore the function of these smaller cells is motor. That the whole section of the brain cortex in this region is motor, it is very difficult to believe, though such a view is apparently supported by some very considerable facts. Finally, as regards experimental evidence, enough has been said on that point to avoid adding to it. The only experiments that will show anything are the observations on human beings. Until after small localised portions of that region have been excised, and a reliably large number of such cases have been thoroughly examined and reported, it will be impossible to do more on this problem than admit its present insolubility.

Professor Haycraft.

I should like to draw attention to a change which has recently taken place in the views of many physiologists respecting the functions of nerve cells and nerve centres. This change is due, I think, to a more philosophical conception of the development of the brain, and to the introduction of improved technical methods for its histological investigation. I venture to make these remarks, because in my opinion the discussion, as to whether certain regions of the cerebral cortex are motor or sensory, can resolve itself into a statement of what they really are in terms of these newer conceptions. We must remember that the central nervous system in higher animals is interposed between the sensory surfaces of the body and the muscular system, etc. and that the nervous connec-

tion between a sensory surface and a muscle forms a loop passing through the brain or cord. In its path this loop is beset with nerve cells, which are spoken of as being sensory or motor cells. As a result of stimulating the skin of an arm, an impulse will pass to the cord and may turn back to a muscle of the same limb. It may, however, make a larger circuit, and pass up to the brain, to the grey matter around the fissure of Rolando, back to the cord, and out to the muscle. In the second case, it passes to the brain, in order that it may be correlated with other impulses passing in other loops belonging to the special senses; for the brain is to be considered strictly as an enlargement due to the presence of these sense organs. There is a greater and greater tendency to look upon the central nervous system as nothing more than a vast mass of communications, whereby both sensory and muscular areas can be brought into relationship, whereby the simple connecting strands between sensory cell and muscle, isolated in the lower, are bound together in higher animals. The discoveries of Exner compel us to look upon that wonderful plexus of medullary fibres which he has demonstrated in the cortex, as more important even than the nerve cells found there. No one could examine a preparation of the cortex, prepared by Weigert's method, without being convinced that the interlacing fibres to be observed, connected ultimately with every muscle and sensory surface of the body, form a mechanism which will account for our possession both of muscular co-ordination and intellectual association. If at one time certain cells in this intercommunicating meshwork were looked upon as the special seat of sensation and others as the seat of motion, it is impossible so to view them now. As a result of the passage of an impulse through the nervous system, we may have muscular movement and we may have sensation, but in the nervous path through which the impulse passes it is not possible to say that one part is more motor than another, or to localise sensation to any given spot. Every portion of the path receives an impulse which originates in a sensory area, and passes ultimately to a muscle. Were we to define a sensory cell merely as one that receives an impulse from a sensory surface, and a motor cell as one that transmits an impulse directly to a muscle, we are at once met with a difficulty. The cells in the cortex are on a loop between the sensory and motor cells of the cord, and to which do they belong? It may be inquired, what are then the uses of these cells? It is probable they act as junctions in the nervous network, as suggested by Dr. Hill. They are trophic, and perhaps they act like collections of combustible material placed on a train of gunpowder. In any case it will be advisable to use the terms

sensory and motor quite tentatively, and preferably to speak of these so-called cortical centres as areas of the nervous system through which pass certain sensori-motor loops, and whose destruction will render impossible the productions of those muscular movements, etc. which had previously depended upon their existence.

DR. MERCIER.

Before entering on this discussion, may I first be permitted to express the sense of incongruity which it arouses? It appears to me that a discussion by the Neurological Society, as to whether or not the idea of movement is a concomitant of the outgoing current, bears a certain analogy to a discussion by the Astronomical Society as to whether or not the earth is round.

Dr. Bastian has complained that there has been shown no *à priori* reason why the generally received doctrine should be preferred to his own, which I with difficulty refrain from calling the geocentric doctrine of neurology. This is a complaint which he ought not to have the opportunity of making again, and for want of a better I will supply him with an argument which is, I hope, not open to objections on the score of deficiency in *à priori* character. The organism exists not alone in space, but in the midst of surroundings. Between it and its surroundings but two actions are possible, viz. action of the surroundings on the organism, and action of the organism on its surroundings. These two actions are of co-ordinate importance. Consciousness cannot exist without both. Life cannot exist without both. Into every manifestation of consciousness and into every manifestation of life both elements enter as necessary constituents. We may separate them in analysis, but in fact they are inseparable. In the constitution of consciousness and of life neither of these elements is before nor after the other, neither is greater nor less than the other. Yet, according to Dr. Bastian, the actions of the environment on the organism are known immediately and directly, while the actions of the organism on the environment are known only mediately and indirectly and subordinately and at second hand.

Much has been said as to whether this or that case has been accurately observed and correctly reported, and as to whether this or that fact may not be capable of a different interpretation from that which Dr. Bastian places on it. I am not concerned to enter on these matters, and I will say at once that I will make Dr. Bastian a present of all his facts. I will take his doctrine as he himself states it, and show, not merely that he has made out no case to be answered, but that he himself explicitly admits the accuracy and truth of the orthodox view.

Dr. Bastian says that these cortical centres are not the *real* motor centres. The real motor centres are in the cord and medulla. But what does he mean by a real motor centre? Those only, it appears, which co-ordinate muscular actions into movements. This alone is what Dr. Bastian calls co-ordination. But this is only one case of co-ordination. The combination of movements into acts is a case of co-ordination, and is effected by centres higher than those in the cord. The combination of acts into operations is a case of co-ordination, and is affected by centres higher than the last. The combination of operations into conduct is a further instance of co-ordination, and is effected by the highest centres of all. Every one of these co-ordinations is a co-ordination of movement, is a motor process, and is effected by motor centres. However, I do not desire to be exacting, and I will admit, for the sake of argument, that the only real motor centres are in the spinal cord.

Dr. Bastian's next point is that the cortical centres are not motor, but sensory. They are not motor, because, as we have just seen, the influence descending from them to the periphery has to act through other centres before producing movement. But is it pretended that sensory impressions on their way to the cortex are not co-ordinated and re-co-ordinated and co-ordinated again? and that in undergoing this process they have not to pass through centre after centre? If the motor centres are not motor because other centres intervene between them and the periphery, then for the same reason the sensory centres are not sensory. However, I will yield this point also, and will grant Dr. Bastian all his data, submit to all his arguments, and show that his conclusions, so far from being adverse to the generally-received doctrine, are in fact entirely in harmony with it and corroborative of it.

Dr. Bastian admits that the centres in dispute are the turning-point of the nerve current. He admits that although they are not motor centres, yet from them issue "volitional motor incitations." These volitional motor incitations descend to the "real" motor centres and set them in action, and hence arise movements. Here I have Dr. Bastian on the horns of a dilemma.

What is it that descends to the motor centres? A volitional motor incitation, (p. 87). A volitional impulse, (p. 54). What is that? Does Dr. Bastian mean that the will sits enthroned in the cortical centres and from thence directs the operations that go on in the brain? Does he mean that the will itself passes down the nerve fibres and discharges the cells of the lower centres? He has expressly repudiated any such absurdity. But if he escapes from this horn of the dilemma it is only to be impaled on the other.

If it be not a state of mind that descends to the cortical from the "real" motor centres, then it must be a physical process, a nervous discharge, or nerve current, or molecular movement of some kind. Now mark: this current is an efferent current; it is an efferent current which really is (p. 86) a motor incitation; that is to say, which results in a movement. And this efferent current is also a "volitional impulse;" that is to say, the nervous action is attended by the mental act of willing. Of willing what? A movement. In other words, this "volitional impulse," which on its physical side is an outgoing current, is on its mental side a volition of movement. Now it cannot be contended for a moment that an idea of movement does not form part of a volition of movement. I cannot will to walk across the room without having an idea of walking across the room. So that, according to Dr. Bastian's own statement, *the idea of movement is a concomitant of the outgoing current*, and this doctrine, which he set himself down to disprove, he has laboriously established beyond all risk of confutation. Like Balaam, the son of Beor, he took up his parable to curse, and he has blessed us altogether.

Dr. de Watteville.

From the discussion that precedes, we gather that those who refuse representation in consciousness to the purely motor, or outgoing, processes of the cortex, are not agreed as to the nature of the Rolandic area. Whilst Dr. Ferrier considers it as the cortical equivalent of the anterior horns of the spinal cord, Dr. Bastian argues for its sensory, or rather receptive, character, and attributes to it *kinæsthetic* functions, implying by this word that the so-called motor convolutions are the end stations of sub-conscious centripetal impulses from the organs of movement.

Between these two views that of Dr. Ross forms a transition. According to him, the large cells of the Rolandic area are truly motor, whilst the small cells are receptive, perhaps even sensory.

I do not propose to enter upon this aspect of the question now before us, for the simple reason, that I scarcely feel competent to discuss a point so full of difficulty. But I accept as more consonant with the facts the view held in common by the distinguished neurologists just named, and shared by many of the leading psychologists abroad, that the physical substrata of volition, of the feelings of effort and movement, are antecedent processes to the final, or "motor" cerebral discharge. Dr. Mercier has just given expression, in terms more lively than forcible, to a diametrically opposite opinion. He calls our position a "geocentric" one. I should have thought that, if anything deserved

this epithet in the matter, it was his own attitude with reference to the progress of our knowledge; for the fact of his having thrown in his lot with the "outgoing" school seems to have blinded him to the consideration that their number is but limited. Wundt's name is constantly bracketed with that of Bain in reference to the views he holds upon this subject; but I must confess that, on searching through the pages of the second edition of his 'Physiological Psychology' for clear evidence as to the German author's actual opinion, I failed to convince myself that full justice is done to him by this constant and close association of names. Be this how it may, however, I am afraid that what Dr. Mercier is pleased to call the "orthodox view," will, like many other orthodoxies in the present day, appear to every impartial mind as standing on its very last legs.

But if any arguments are required beyond those already adduced, to show that our conscious life is closely bound up to the activities of our sensory cerebral mechanisms, we might appeal to the fact that consciousness is less an end in itself, than a means towards further ends. It accompanies and appears to guide imperfect action; it tends to disappear with the development of more perfect mechanisms. The main function of sensory centres is to educate motor co-ordinations, the essence of which is their automaticity. Moreover, if discharges of the motor elements were really the substrata of volitions, of the feeling of effort, then we must assume a very deep separation between the mental representation of, and the wish to perform, a certain movement, on the one hand, and the fiat, or volitional act, on the other. Psychological analysis does not seem to me to admit of such a separation. Again, on the principle of differentiation of functions which pervades the evolution of organs throughout the animal series, we have a right to assume that the physical processes, of which consciousness is the subjective equivalent, tend to become more and more localised in circumscribed areas of the nervous system. Hence there is no *à priori* objection to the view which localises them in the receptive areas of the cortex. And in presence of an undoubted law of nature the speculative views of transcendental brain-physiology, such as the theory of the representation (whatever this term may mean) of every part of the body in every part of the cortex, must obviously recede into the background.

Indeed we are not so much concerned here with general arguments and *à priori* considerations as with the evidence of experimental facts. One of the main questions is whether destruction of the Rolandic or "motor" region of the human brain is or is not characterised by a diminution of muscular sense in the parts

paralysed; whilst another is whether such destruction is characterised by a concomitant loss of certain assumed psychical equivalents. Now, leaving for further investigation the former problem, it will be found that the facts at our disposal for the solution of the latter are but few; but, so far as they go, they certainly seem to support the view, that the activity of these centres is not largely if at all represented in consciousness. Lesions, at any rate unilateral lesions, strictly localised in the ascending convolutions, do not appear to be accompanied with more than feeling of mechanical inability to move the corresponding limbs.

The attempt to identify discharges of motor cells, or the "outgoing current," with the physical process of volition, involves a serious difficulty; for if it were so, every reflex process including such a discharge would thus, *ipso facto*, become a voluntary act. Nor do I understand why, if the subjective phenomena attending volition to move a limb after amputation (spectral hand) are mental equivalents of the motor cerebral discharge, similar phenomena do not accompany similar discharges in other cases, where the movement is similarly willed but not carried out—in spinal paralysis, for instance. This fact clearly points to the lesion of the sensory nerves, and consequent irritation of the sensory centres, as the probable cause of these hallucinations.

When I try to analyse the contents of my consciousness during the performance of a deliberately willed movement, I find myself in presence of a highly complex state, in which the senses of sight and touch enter for a very large share. I cannot really separate the visual representation of the moving limb from its numerous tactile accompaniments, such as contacts with clothes, air, surrounding objects, or other parts of the body. These elements, which form a large portion of what is called the "feeling of movement," depend obviously upon sensory activities of the brain.

But there is also involved in the state of consciousness described as the volition of a movement, the phenomenon of *effort*, which is itself reducible to simpler elements. It includes numerous afferent feelings; sensations of cutaneous and deep pressure; of strain on the muscles, tendons, and ligaments, not always confined to the limb directly involved, but often diffused over a larger area; there is also a sense of air pent up in the lungs so as to give a firm support to the thorax, of excited heart action, of throbbing arteries and venous congestion. Sooner or later the sense of spent energy, of fatigue, supervenes. All these elements may be accounted for by purely centripetal impressions. But in addition there is the distinct consciousness that I am engaged in the effort, that I am the cause of my movement. Now psycho-

logically it might be maintained that I have reached cognizance of my own self as a cause through a process of inference; but this is not the place to enter upon such considerations. My object is to show how those who uphold the outgoing theory, and who object to the afferent doctrine on the ground of its supposed inability to account for this possibly primary datum of consciousness, may get over their difficulty and frame a hypothesis that does not postulate more than the activities of the receptive or sensory portions of the cortex as substrata of mental states generally.

Assuming that consciousness is the mental equivalent of the metabolic processes attending the activity of certain cortical elements, one might expect certain subjective states to accompany the molecular changes in the cell considered as a *terminus*, as it were, and as storing up impressions from the periphery; and other states to accompany the action of the same cell considered as a *switch*, or a *relay* for the further transmission of force. A physical basis might thus be found for dynamic as well as static conscious states, localised in the purely sensory regions of the cortex. And as will is, after all, but intellect in action—intellect itself being but the elaborated product of experience—it is not unnatural to look for the physical substratum of both cognitive and volitional phenomena in the same organs, viz. in the receptive and higher elaborative layers of the sensory cerebral sphere. Among the latter I include those tracts of cortical substance that preside over the complex inhibitions in which we must seek the physical basis of the psychological illusion described by introspective observers as the "liberty of the will," or the faculty of selection of the motives in human actions.

Whether this theory is sufficient to account for all the aspects of the dynamic states of consciousness (including the feeling of fatigue alluded to by one of the previous speakers), is not material to the present argument. It is at least as plausible as that which postulates the activity of purely motor-cortical elements as the embodiment of our mental states in voluntary movements. It is suggested here merely as an alternative hypothesis for the consideration of the supporters of the "outgoing" theory of the muscular sense. Neither they nor their opponents have as yet, so far as I know, considered discharges of *sensory* elements in the light in which I have endeavoured to place them. Should the attempt lead to nothing but a refutation, I shall yet congratulate myself on the opportunity afforded to elicit further discussion of a subject as important as it is interesting.

REPLY OF DR. CHARLTON BASTIAN.

The task I have undertaken is, as I am fully conscious, one of extreme difficulty. I have endeavoured to show that a view widely accepted in this and in other countries, to the effect that there are motor centres in the cerebral cortex specially concerned with the execution of voluntary movements, is one which, if not actually erroneous, is, at least, not at all more probable than a counter view, which maintains that these so-called motor centres of the cortex are, in reality, sensory centres constituting the termini for "muscular sense" impressions. It has been through the experiments and writings of Dr. Ferrier, in the main, that the former view has gained so wide an acceptance in this country. Being strongly inclined to adopt the above-mentioned counter interpretation, as one capable of affording a much better explanation of known facts, it became of great importance for me to scrutinise very carefully all the steps by the aid of which Dr. Ferrier had arrived at his conclusions. It was, however, equally important, looking to the difficulty of my task, that full liberty should be accorded to me candidly to point out what I might consider to be defects, contradictions, or insufficiently warranted statements of any kind in the facts or arguments by which the opposite doctrine has been supported. Without this licence having been granted to me, no progress could have been made in dealing with so complicated a subject. Whilst deeply grateful for the consideration and attention which the Society has already accorded to me, I fear I must now crave no small measure of further indulgence.

It may be useful for me at this stage briefly to recapitulate some of the results of my scrutiny, and some of the points to which I have desired to call especial attention.

(1.) I was compelled to point out that my scrutiny of Dr. Ferrier's writings, bearing on this subject, led to the conviction that his doctrines did not hang together at all well; that they were even made up of contradictory statements.

(2.) I also came to the conclusion, that some of his fundamental positions, supposed to be based upon clinical evidence, were in reality faulty, being drawn from too narrow a view of altogether exceptional cases, or else resting upon inadequate observation and inquiry.

(3.) I had to contend that experiments upon animals were quite useless for throwing light upon the question of the situation of the centres for muscular sense, and that all previous conclusions thus arrived at were valueless; that we must, instead, look minutely into the effects of cortical disease, and, better still, into the effects of excision of portions of the cortex in man, in order to ascertain whether or not lesions in the so-called motor area are attended with any corresponding loss of muscular sense.

(4.) Further, I stated my belief that Dr. Ferrier seemed not to have comprehended the all-important difference between the symptoms that would result from a mere cutting across of the afferent tracks for muscular sense impressions, and those that would result from destruction of the cortical termini for such impressions.

(5.) I endeavoured to show that Dr. Ferrier had taken an inadequate and indeed erroneous conception of the functional importance of kinæsthetic centres, and that upon them, in reality, would devolve just such functions as those which he supposes to be carried out in cortical motor centres.

(6.) I maintained that neither on physiological nor on psychological grounds was it needful to postulate the existence of motor centres in the cortex.

(7.) I also argued that the doctrine of the existence of motor centres in the cortex would probably never have been heard of had it not been for the importance previously attached to a doctrine now disproved, viz. that of Wundt and Bain, to the effect that our feelings of effort are due to psychical processes directly correlated with the activity of motor centres—a doctrine which Dr. Ferrier absolutely rejects, though it is still held, and has always been maintained by Dr. Hughlings-Jackson, the original propounder of the notion that motor centres exist in the cerebral cortex.

All that Dr. Ferrier has said in reply will be found to have reference to some of the points which I have referred to under the first three headings above mentioned.

In support of my position, that the doctrines upon which Dr. Ferrier originally based his conclusion that motor centres existed in the cortex were mutually contradictory, I called attention, as far back as 1880,[1] to the fact that whilst in chap. ix. of his 'Functions of the Brain' he thoroughly adopted and did much to establish the view, that our sense of movement was altogether dependent upon centripetal impressions, strangely enough when

[1] 'The Brain as an Organ of Mind,' 1st ed. p. 569.

he came to chap. xi. he expressed himself in the most emphatic manner as if he had still been an adherent of that doctrine of Wundt and Bain which he had already so successfully combated.

In the second edition of the 'Functions of the Brain,' without comment or explanation, the expressions last referred to were radically altered. Yet Dr. Ferrier seems to be unaware of it. He now says, "I have made no change of front, and am glad that in the second edition of my work I have succeeded in clearing away any misconception that may have existed as to my real meaning, which is, that I consider that the activity of motor centres and motor nerves by themselves is entirely outside the sphere of consciousness." Now let us see how far Dr. Ferrier can really claim to have been the pioneer in 1876 of that doctrine which I clearly enunciated in 1880—by placing side by side an original passage, and the same passage altered as it now stands in his second edition.

'Functions of the Brain,'
1st Edition, p. 266.

"In the same manner as the sensory centres form the organic basis of the memory of sensory impressions, and the seat of their representation or revival in idea, so the motor centres of the hemispheres, besides being the centres of differentiated movements, are also the organic basis of the memory of the corresponding movements, and the seat of their re-execution or ideal reproduction. We have thus a sensory memory and a motor memory, sensory ideas and motor ideas; sensory ideas being revived sensations, motor ideas being revived or ideal movements. Ideal movements form no less an important element in our mental processes than ideally revived sensations."

'Functions of the Brain,'
2nd Edition, p. 436.

"In the same manner as the sensory centres are the organs of special sensory perception, and the organic basis of the memory of sensory impressions, so the motor centres of the cortex, besides being the organs of differentiated movements, are the organic basis of motor acquisitions. But while the activity of the sensory centres reveals itself subjectively in consciousness as ideation or feeling, the activity of the motor centres has no subjective side apart from the functioning of the sensory centres, with which they are associated. Modifications of consciousness are correlated exclusively with the functioning of the sensory centres of the cortex."

It seems to me difficult to imagine any more complete change of front than is represented by these two passages, and if Dr. Ferrier does not admit it, I am afraid all will consider him hard to be convinced.

But let us turn to another point. In support of his fundamental statement, that "co-ordination is possible in the entire absence of any sense of movement," Dr. Ferrier can still only refer to one very exceptional case, that of Remigius Lens, which does seem, as I have already admitted (p. 15), whatever the explanation

may be, to lend support to his statement. The rule, however, is, as I contend, that the loss of muscular sense distinctly impairs the power of accurately co-ordinating movements when the eyes are closed. I, of course, do not mean to say that inco-ordination of movement may not be brought about also in other ways, and from what Dr. Ferrier says it would seem to be needful to remind him of this. Again, I maintain that frequently in cases of hemi-anæsthesia there is no loss of muscular sense, and no inco-ordination of movement; but that in a much smaller proportion of cases there is, in addition to the cutaneous anæsthesia, a profound loss of muscular sense (pp. 11–22). Dr. Ferrier says he is "amazed" at these statements, and adds, "I do not know a single observer, with the exception of Dr. Bastian, who has ever seen a case of hemianæsthesia in which muscular sense was not abolished or impaired in proportion to the degree of the anæsthesia existent." After what Dr. Ferrier has heard from Dr. Ross this evening, he will, at all events, have learned that the experience of that distinguished observer is in accordance with my own. If time and space permitted I could easily quote the observations of several other distinguished physicians to the same effect. I trust, however, that two or three such quotations may suffice to convince Dr. Ferrier that it is his own view and not mine which is most calculated to excite surprise in the minds of others.

Dr. Russell Reynolds has made the following remarks[1] concerning cases of marked hemianæsthesia under the care of M. Charcot, which he had had the opportunity of examining for himself:—"There was no deficiency of knowledge as to the position of the limbs; but M. Charcot informed me that he had met with this deficiency in other cases." Again, Dr. Arthur Gamgee writing[2] concerning hystero-epileptic patients whom he had examined in M. Charcot's wards, speaks in the following terms of this affection:—"It is associated with hyperæsthesia in one or both ovarian regions, and is usually attended by hemianæsthesia, and more rarely by anæsthesia of both sides of the body. There is some, if not complete, loss of tactile sensibility, and usually absolute insensibility to pain (analgesia) of skin and all other sensitive structures on the affected side; the muscular sense being, however, nearly always preserved." Let it not be supposed, however, that the recognition of differences in the degree of hemianæsthesia of the kind to which I have referred, is a result only of modern observation. I would call Dr. Ferrier's attention to the fact, that the view opposed to his own was clearly substantiated by

[1] 'Lancet,' May 12, 1877, p. 679.

[2] 'Brit. Med. Jour.,' Oct. 12, 1878, p. 545.

Duchenne more than thirty years ago. In his work 'De l'electrisation localisée' (p. 410) this accurate observer calls attention to the existence of two categories of hemianæsthesia; one (*a*) in which there is loss only of cutaneous sensibility, and another (*b*) in which there is, in addition, loss of the sensibility of the muscles, bones, and nerves. In regard to representatives of the former category, he says:—"Ils ont la conscience des mouvements mécaniques qu'on leur imprime, de l'étendue des mouvements qu'ils exécutent eux-mêmes, de la pesanteur, de la résistance; chez eux, enfin, la contractilité volontaire n'éprouve aucun trouble dans son action physiologique." In his description, however, of a case belonging to the second category he says:—"Quand il est dans l'obscurité, ou si on l'empêche de voir, il n'a pas la conscience de la position de ce membre, ni des mouvements les plus brusques qu'on leur imprime." He then goes on to point out that this second category includes two sub-varieties, in one of which (1) the patients are quite able to execute movements when their eyes are closed, though they have no consciousness of the extent of such movements and are unable to estimate weight or resistance; whilst in the second sub-variety (2) the patients (under similar conditions), though presenting the defects abovementioned, lose also their power of executing even the simplest voluntary movement. Of the latter class I have quoted three very striking examples, recorded respectively by Duchenne, Briquet, and Bazire (pp. 16–20).[1]

[1] Since Duchenne's time no one has attempted to give an explanation of these remarkable cases excepting that which I have suggested (p. 36). I have supposed that in them we have to do with a functional defect (that is a state of lowered irritability) in the cortical termini for "muscular sense" impressions, associated with some defect in the functional integrity of the afferent channels for such impressions, as they pass through the posterior part of the internal capsule. According to Duchenne, it was the absence of what he termed "la conscience musculaire" which determined the inability to perform the simplest voluntary movements in this class of cases (*loc. cit.* p. 414). Now it seems clear that the revived activity of kinæsthetic centres, such as occurs in the conception of a movement, would exactly correspond in the time of its operation with what Duchenne termed "la conscience musculaire," which he says "dans l'acte des mouvements volontaires, semble précéder et déterminer la contraction." Duchenne found that the missing power in his cases might be restored by very strong faradisation of the muscles (which sufficed to restore sensibility to them) even though the cutaneous anæsthesia persisted. At the same time that sensibility was thus restored to the muscles it is easy to imagine that the powerful peripheral stimulation (faradisation), acting upon the sensory nerves of muscles, might easily have sufficed to rouse their previously dormant cortical termini into activity, and thus may we account for the restored ability to perform movements when the eyes are closed. Thus, also, may be explained the frequent similarly good effects of powerful faradisation of the affected muscles in some cases of hysterical

Another firmly rooted belief held by Dr. Ferrier is the converse of that last referred to, viz. that cases are never met with in which muscular sense is lost apart from loss of tactile sensibility, or altogether out of proportion to the loss of tactile sensibility. I fully admit that few definite records of such conditions exist, but I am far from believing that they will not be found when they are carefully looked for. Thus, I think it may be possible to induce such a condition in part artificially, by dealing with a case of functional hemianæsthesia of the second category, after the fashion adopted by Duchenne, as referred to in the note on the previous page—that is, by strongly faradising the skin so as to restore sensibility to it whilst the muscles are this time left in their anæsthetic condition. Though Duchenne says he has several times done this, he has left no specific statements on record as to the continued existence of loss of knowledge of the position of the limbs, and of their passive movements, in such cases. He does, however, state that in the instances of the second sub-variety in which he has performed this experiment, the patient's inability to move the limbs voluntarily without the aid of sight impressions has persisted when sensibility has been restored to the skin, so that it becomes highly probable, to me at least, that the loss of so-called muscular sense would also have persisted under these same conditions.

In illustration of this kind of defect I have, however, in my paper suggested that we should search for it under a different set of conditions—that is, in cases of a wholly different type. The conducting tracks for cutaneous and for muscular impressions seem to be more or less separate from one another, though contiguous in their passage through the internal capsule, but I suggest that they proceed, in the main, to entirely separate cortical stations. My provisional hypothesis, in fact, is that the convolutions comprising the so-called "motor area" are in reality the cortical termini for muscular sense impressions. On this point, as I fully admit, evidence in actual proof has yet to be obtained, though I have attempted to show how well this hypothesis is capable of explaining known facts. As strongly bearing upon this problem, I contend that observations on the lower animals are worthless for the settlement of the location of this endowment. We must look to cases of cortical disease in man, and especially to cases in which, owing to disease, the so-called arm and leg centres (but especially the former, on account of the

paralysis—a condition which may be supposed to depend, as I have suggested (p. [illegible]), upon still lower conditions of functional irritability in corresponding muscular sense centres.

greater facility with which defects of muscular sense may be detected in the arm and hand) have been excised by the surgeon.

With this object I quoted a few details of two cases briefly reported by that excellent observer Landry (who had made defects of sensibility of different kinds a special study), in which, he says, there was marked defect of muscular sense, and but little, if any, defect of cutaneous sensibility, as possible cases (looking to all that is said concerning them) of cortical disease in the Rolandic area. Similarly, as still more suggestive, I cited a case recorded by P. Zenner (which was kindly brought to my notice by Dr. de Watteville); and, as amounting to something more like proof, so far as they went, I also cited two cases of excision of portions of the cortex which had been recorded by Mr. Horsley. One of these latter patients was in part under the care of Dr. Ferrier himself, and, as we have heard, he thinks nothing of the evidence either of them may afford in support of my hypothesis. But after weighing all the doubtful elements which they presented, Mr. Horsley is not of that opinion. He has expressed, indeed, to-night the same kind of conclusion that I have drawn when he says:—"I would not rely on their evidence further than to regard them as tending to show that the so-called motor region is the centre of muscular sense."

Dr. Ferrier, however, refers to another case of operation for disease in the right Rolandic area by Mr. Horsley, which seems to tell against the above-mentioned conclusion. I had not the opportunity of seeing the patient in question myself, who is referred to by Dr. Ferrier as constituting "a crucial experiment, which absolutely knocks the ground from under Dr. Bastian's feet." It may well be imagined, however, that this case was examined and considered most carefully in all its aspects by Mr. Horsley, and the terms in which he has spoken this evening concerning the man, referred to in such positive language by Dr. Ferrier, are altogether different. He says:—"The whole surroundings of the case are so very complicated that even after the death of the patient I should not like to adduce it alone in support of an argument one way or the other."

I adduce the cases above referred to only for what they are worth. We must undoubtedly wait and seek further evidence. I feel this necessity fully, though I hope to have made out a sufficiently strong case to lend an increased stimulus to the minute observation of cases of disease in, or removal of, parts of the excitable area of the cortex. Certain it is that Dr. Ferrier's views do not lend any encouragement whatsoever to the notion that there would be loss or diminution of

muscular sense from disease in, or excision of, these regions of the cortex. While, on the other hand, if my hypothesis be true, it follows, as a necessary consequence, that in such cases there should be evidence of some loss or defect of muscular sense (see p. 73) in association with motor paralysis. There might or might not be, in addition, a certain amount of loss of tactile sensibility, such as was present in the case of Zenner. Such a combination might be met with if all tactile impressions do not proceed, as Dr. Ferrier supposes, to the falciform lobe as their cortical terminus. That is, in my opinion, also a point which requires to be carefully looked to in the future, especially in view of the statements made by Munk.

Dr. Ferrier reiterates his belief, that the cortical centres in the excitable area are true motor centres; but he does not adduce a single reason tending to displace my contention, that all known facts can, as I have endeavoured to show (pp. 80–84), be equally well explained in accordance with my hypothesis. Nor does he attempt to controvert any other of the positions which I have supported.

I am happy to find that between Dr. Ross and myself there are many points of agreement. Thus, he is at one with Dr. Ferrier and myself in regard to the fundamental point, that the sense of effort or of resistance is correlated with ingoing currents and the activity of sensory centres; he agrees with me, in opposition to Dr. Ferrier, that in the majority of cases of hemianæsthesia there is no correlative loss of the "muscular sense" endowment; he is further in agreement with me (and with Dr. Ferrier's later view) in regard to the absence of psychical states in association with the activity of motor centres. There is additional agreement between us in regard to another point, on which Dr. Ross says, "I at least give my unhesitating adhesion to Dr. Bastian's opinion when he says that the kinæsthetic centre is the last centre which must be excited anterior to the movement in any psychical action." There is even a readiness on the part of Dr. Ross to find that the kinæsthetic centres are situated where I believe the most important part of them to be located, viz. in the so-called excitable area of the cortex, since he says :—"I think it, however, quite likely that the kinæsthetic and motor centres coincide, in so far as that the former are situated in the two outer and the latter in the third layer of the cortical cells of the parieto-frontal area of the cortex."

One most fundamental difference, however, separates the views of Dr. Ross from my own. I read with genuine surprise the statement in which he says :—"I do not believe that any amount of repetition tends to convert a movement which was once

voluntary into a reflex action either in the individual or the race." Yet a further study of all that he says concerning movements has shown me that this statement (apparently so opposed to the philosophy of Evolution in which Dr. Ross is a firm believer) is due to what I take the liberty of terming arbitrary limitations that he has chosen to give to the words "voluntary" and "reflex" respectively. I am by no means convinced that I have been so very wrong in making use of commonly accepted terms, or that Dr. Ross is so right as he imagines in his use of his own terms and in his classification of movements. This, however, is a side path, into which I will not be seduced, even in order to defend myself from the strictures of Dr. Ross. Elsewhere I have pointed out more in detail what meanings I attach to the different terms commonly used in the classification of movements,[1] and I am not aware that the least ambiguity has crept into my argument on the present occasion from the use which I have made of these terms.

A view has been broached by various persons of late to the effect, that the excitable area contains both sensory and motor centres, rather than motor centres only, as Ferrier contends, or sensory centres only, as I am more inclined to maintain. This view has to-night been mentioned favourably both by Dr. Ross and Mr. Horsley. They are not indisposed to believe that the upper small-cell strata of the Rolandic convolutions may be concerned with sensory functions of the kinæsthetic type, though both of them seem strongly wedded to the notion, that the large cells in the deep layer are real motor cells—in the main because of their complete organisation, large size, and analogies with motor cells in the anterior cornua of the spinal cord. I must confess I do not sympathise with this notion, though I do not by any means regard it as an essential part of my hypothesis, that the whole depth of grey matter in the excitable area should be given over to kinæsthetic functions. Some evidence already in our possession tends to show that tactile sensibility may also be, to some extent, represented in this area; and should this evidence be strengthened, I shall be quite prepared to believe that the upper layers of the cortex of the Rolandic area are concerned with the reception and revival of these impressions, while the lower layers are concerned with the reception and revival of kinæsthetic impressions. I am not influenced by the mere fact of the large size and elaborate structure of the cells of the deep layer, as Dr. Ross and Mr. Horsley seem to be, and that for two sets of reasons. First, I fail to see any necessity for the existence of motor cells in the cerebral cortex, because I believe that volitional action, so far as it is dependent

[1] 'Brain as an Organ of Mind,' p. 459.

upon cortical substrata, is essentially intellectual action, in which the activities of motor cells take no direct share. On this part of the subject I shall have something to say further on. Neither Dr. Ross nor Mr. Horsley adduçes anything, from this point of view, in support of a necessity (which possibly they may feel) for postulating the existence of motor centres in the cerebral cortex; they dwell rather upon the histological analogies existing between the large cells of the cortex and those of the motor regions of the spinal cord. But this second set of reasons has no cogency for me. I mistrust such analogies altogether, knowing how deceitful they have often proved. Then, again, I think it not improbable that the large size and marked development of these cells may be as much a necessity for them (in view of the kind of functions I suppose them to subserve), as for the motor cells in the anterior cornua of the cord. It should, further, be remembered by Dr. Ross that, in accordance with my view, the psychical states associated with the activity of these cells are non-vivid, vague and comparatively faint, and that if there are (as I suppose) no motor cells in the cortex, the large cells in question there situated would, as constituents of kinæsthetic centres, have functions to perform of a kind wholly different from those of the constituents of all other cortical sensory centres; since it would be from them alone that efferent incitations would pass over from the cortex for the excitation of motor centres of whatsoever grade. Thus it would happen that in many cases molecular movements would have to be transmitted from different groups of these kinæsthetic cells, now to the cervical, now to the dorsal, and now to the lumbar regions of the spinal cord, through sets of internuncial fibres correspondingly increasing in length. This kind of function may well necessitate a cell of large size; so that I cannot feel that the argument from mere histological characters carries any weight with it, or can be said, when looked at more closely, to tell more strongly in favour of the one than of the other hypothesis.

Dr. Ross says:—"The main question which divides Dr. Bastian from Dr. Ferrier is the point at which the centripetal conducting paths and centres cease and the centrifugal paths and centres begin. Most of us will at once say that this point is to be found in the large caudate, pyramidal cells of the third layer of the cortex." That is not, to my mind, an adequate statement of our difference; else it would not be possible for me to say, as I do, in reply to Dr. Ross,—I also believe these cells to be the point where centripetal paths and centres cease, and where centrifugal paths begin—if it be permissible to consider as centrifugal the path which connects sensory with motor elements in an ordinary sensori-motor unit.

Mr. Sully, Sir James Crichton-Browne, the President, and Dr. Mercier all more or less strongly support the notion of Bain and Wundt, that psychical processes are directly associated with the activity of motor centres and nerves, and that it is in and through them, in the main, that we derive our notions of weight and resistance.

Now it was because I believed this view to have been absolutely disproved, that I devoted comparatively little attention to it in my paper. It seems to me, as I pointed out long ago, that the cases of hemianæsthesia of Duchenne's second category (pp. 11–20) suffice of themselves completely to displace this doctrine. Referring to the case of Demeaux, I then said[1] :—"There was in this woman a total disappearance of all that kind of knowledge which has, by one or other, been ascribed to, or supposed to be derived from, the 'muscular sense.' The woman was ignorant of the position of her limbs and unconscious of any movements that she might execute. The volitional centres, the spinal motor centres, the motor nerves, and the muscles were capable of being called into activity as before—yet all the information supposed to be derived through the 'muscular sense' had vanished." . . . . "No clearer evidence than this, together with what has been previously mentioned, could be forthcoming to show that the knowledge of the position of our limbs, of their movements, and of the state and degrees of contraction of our muscles generally, does not depend, as Wundt, Bain, and others assume, upon impressions that are 'concomitants of,' or that coincide with, 'the outgoing stream of nervous energy.'"

But if anything further be needed to convince those who still have a lingering affection for this doctrine above referred to, other strong facts in condemnation will be found in Dr. Ferrier's 'Functions of the Brain,' and in Dr. James's memoir on 'The Sense of Effort.'

All that Mr. Sully says concerning the hallucinations that are often known to follow the loss of a limb, can be fully explained by reference to the irritation of sensory nerves and sensory centres (pp. 37–40). That this is so derives strong confirmation, as Dr. de Watteville points out, from the fact of the total absence of any such hallucinations in cases of complete anterior poliomyelitis, where what is practically a maiming occurs only on the motor and not at all on the sensory side of affected limbs.

Nor can I think that any real light will be thrown upon the fundamental problem by mere speculations concerning the very doubtful pathogenesis of "fatigue and fidgets." I will not attempt, therefore, at any length to follow Sir James Crichton-Browne in

[1] 'Brain as an Organ of Mind,' 1880, p. 700.

his speculations upon these subjects. Still it may be remarked that the "fact of the indisposition for mental as well as for bodily exertion" under conditions of fatigue would of itself seem to point to some general modification in the great nerve centres, and not to an altered molecular condition only of hypothetical "motor centres" in the cortex. Similarly indecisive, as it seems to me, is his supposition in regard to the causation of fidgets, which he imagines to be due to a "surcharged or excitable state of the same centres." It is indecisive, I mean, for helping us to gauge the relative correctness of his view and of mine in regard to the true functions of these particular cortical centres, seeing that it would be equally open to me (holding the views which I have brought forward as to the functions of these centres) to explain with the same degree of plausibility the phenomena of fidgets by the phrase above quoted, adduced though it was by Sir James Crichton-Browne in support of the rival doctrine.

I believe, however, that such speculations are too vague to be of any service to either of us. When Sir James Crichton-Browne says, "I cannot divest myself of the belief that as we have perception at the end of ingoing currents, we must have apprehension at the beginning of outgoing currents," I see in that statement the enunciation of a favourite belief rather than evidence in support of its truth.

Again, I am afraid, Sir James Crichton-Browne has read my paper to little purpose if he supposes that I am willing to assent to his notion, that to endow the convolutions of the "excitable area" with kinæsthetic rather than with motor functions is "to degrade a large region of the cerebrum from its high estate, and leave it a mere superfluous intrusion in the brain mass;" or if he imagines that it forms any part of my view to say that kinæsthetic centres have "no part to play in ideation," or that they are, in any way, "redundant or superfluous." In regard to the performance of voluntary movements, Sir James Crichton-Browne says, "Sensory impressions from the part to be moved can play no part. They come too late." That is true. But what I contend is, that it is the molecular motions concerned with the memory of past visual and kinæsthetic sensations occasioned by similar movement, whose revival takes part in the production of the movement now about to be executed. These centres are, in fact, those concerned with what he terms "the regulation of the jet," and that, through the intervention, in part, of revived impressions, and, in part, of now occurring impressions occasioned by present movements.

Dr. Hughlings-Jackson, in the main, reiterates his well-known views, but unfortunately, in spite of them, or rather after

having given them the fullest consideration, I have felt compelled to come to the very opposite conclusions. He does not in any way endeavour to show me where I am wrong, or attempt to explain away the difficulties which, as I have pointed out, now make his favourite doctrine untenable. I am as fully imbued, perhaps, as he is with the great value of the doctrine of Evolution, and have been so for the last five-and-twenty years at least. I agree with him further, that "the unit of constitution of the nervous system is a sensori-motor unit." But I can neither join with him in supposing that we have any knowledge whatsoever of psychical accompaniments of the molecular processes occurring in motor centres; nor that there is the least need, in accordance with the doctrines of Evolution, for supposing the existence of motor centres in the cerebral cortex. Much of that knowledge which he ascribes to molecular movements taking place in motor centres, I believe may be better accounted for by molecular movements taking place in sensory centres. The activity of sensory centres constitutes in all cases, as I maintain, the necessary prelude to, and the guidance by which, this or that kind of activity is to be awakened in the constituent elements of motor centres, for the production of purposive movements. Dr. Hughlings-Jackson says:—"I contend that when I make a movement with my hand I have a psychical state just as certainly as I have another when I see that red chair." In this I quite agree with him, only I say that the psychical state is compound in its origin, and of the kinæsthetic order. But when he says, "I submit that we have psychical states attending activities of motor elements," I do not at all agree with him, seeing that he has left unanswered all the overwhelming objections to this doctrine—and especially if the assertion which immediately follows is to be taken as a type of the only evidence he can adduce in its support. What he subsequently adds is, in my opinion, of equally doubtful value. He says:—"For these psychical states there is no name except in the case of words; words are psychical states attending activities of motor elements representing certain very special and complex articulatory movements." But to make such a statement without proof as a contribution to this discussion, when Dr. Jackson knows that it is a view directly repudiated by myself and many others as an appanage of the above-mentioned disproved doctrine, is no more likely to weaken the tenability of my view than to strengthen his own. Merely to reiterate such statements is surely useless when the underlying doctrine is itself fast dying if not actually dead.

Again, how what Dr. Jackson says touching the errors of the

"popular psychologist" can be supposed to throw light on this discussion I fail to understand, as I do not know whether or not he imputes to any one who has taken part in it the crass absurdities which he mentions.

I find it a little difficult fully to appreciate the intended cleverness of Dr. Mercier's contribution to this discussion, because it at once impresses me with a sense of its hollowness. Like a bubble, it only requires to be pricked to vanish into thin air.

Dr. Mercier is supremely indifferent as to facts for the establishment of his own views, and correspondingly inaccurate in his interpretation of my views. He professes to define my notion as to what constitutes a real motor centre, and also to state what I mean by co-ordination, though in neither case is the result such as I can accept as a statement of my view. He seems to think his own notion that all co-ordination of movements occurs in motor centres of higher and higher rank, from the cord upwards to the cerebral cortex, must necessarily be true. Here, however, I venture to differ from him in two respects. First of all, I do not accept as motor his motor centres of the cortex; and, secondly, I say that co-ordination of movements mainly takes place in, or is brought about by, different sensory centres, of higher and higher rank and endowed with more and more complexity of function, the lowest being in the spinal cord and the highest in the cerebral cortex. Further, I am of opinion that it is under the influence of these sensory centres of different grades that motor elements in different motor centres are called into simultaneous or successive activity, as a result of which connections become established between them (motor co-ordinations), with the effect that subsequent movements of the same kind are rendered more and more easy. Co-ordination is, therefore, in my opinion, primarily and essentially dependent upon the functional activity of sensory centres, though the motor centres respond secondarily by the establishment of intercellular and intercentric connections.

Again, the reason which Dr. Mercier imputes to me as the support for my belief, that the centres of the "excitable area" are not motor, is not in the least in accordance with what I say. I have sought, on the one hand, to bring forward some evidence tending to show that these centres are the termini for a certain class of sensory impressions, and on the other, that there is no need for postulating the existence of cortical motor centres at all.

After such various statements of his own and not too accurate representations of my views, Dr. Mercier approaches the climax of his argument not without signs of exultation. He triumphantly proceeds to show how, in his opinion, I have performed the

remarkable feat of establishing in an incontestable manner the doctrine which I sought to refute. Here again he must permit me to differ from him. I am seemingly led to this inane result by dint of a little jugglery on the part of Dr. Mercier with my words, coupled with a *suppressio veri* which is equally disingenuous. Thus, by attributing to me the view, that the efferent current from the "excitable area" is attended by an act of willing, he seeks to make me say that "what on its physical side is an outgoing current, is on its mental side a volition of movement," whereas I never said anything of the kind. My contention is that the act of willing immediately precedes the outgoing current, and that the molecular movements which constitute this current are in part engendered in, and receive their final direction from, the functional activity of *sensory* centres of kinæsthetic type entering into the composition of the excitable area of the cortex.

Now what Dr. Mercier erroneously terms the commonly received doctrine (i.e. the disproved doctrine of Bain and Wundt) is, that some of the sensations accompanying movements are concomitants of the outgoing current from *motor* centres through whose activity they are realised in consciousness; whereas my contention is that all the sensations accompanying and occasioned by movements are realised, through the intermediation of sensory nerves, in sensory centres. Yet Dr. Mercier is pleased to say that I have proved the very doctrine I set myself to controvert. I fancy few will be found to agree with him.

The remarks of Professor Haycraft are interesting, and it is not difficult to recognise in them the expressions of a genuine seeker after light. When he says that a sensory impulse from the arm "passes to the brain in order that it may be correlated with other impulses passing in other loops belonging to the special senses; for the brain is to be considered strictly as an enlargement due to the presence of those sense organs," I am quite prepared to agree with him. Much of what follows, however, seems to me to be either obscure or of doubtful cogency. I do not think we can accept his definitions of a sensory cell and of a motor cell, since they would apply, for the most part, only to certain cells in the spinal cord or medulla. But whenever an impression of a sensory order ascends through the spinal cord to the brain, it may and probably does pass through two or three sensory centres, in which it may be brought into possible correlation with other afferent impressions, before reaching certain groups of cells in the cortex. Now these latter would seem to be typical sensory cells, since their activity immediately lights up a correlated activity in many

parts of the brain, the whole being attended by the development of certain psychical states corresponding with sensory and perceptive processes. But the activity thus excited may extend in still wider and more complicated circles throughout the brain, into regions which constitute (if we may so speak without implying anything like topographical exclusiveness) annexes of the sensory centres. The combined simultaneous activity of large portions of the whole of these brain areas would correspond with definite intellectual exercises such as the formation of general notions and concepts, together with abstract reasoning generally. All these portions of the brain must, however, be in the closest functional relation with those portions of the auditory, visual, and kinæsthetic centres in which corresponding impressions connected with words in their auditory, visual, and kinæsthetic relations have been organised. By this kind of association language and the power of thinking are mutually strengthened, and undergo a co-ordinate development.

Thus, here in the cerebral cortex, as a whole, we have the birthplace and home of man's intellectual, moral, and emotional nature—the physical substrata being cells of sensory type and others more or less modified, together with the inter-communicating networks of all kinds by which those cells are brought into relation with one another.

But, it may be said, is there not a part of the cortex devoted to volitional action, and does not this part contain motor centres and motor cells? This is a view which is popular at present, and it is the position which Dr. Ferrier, following the lead of our President, has done his best to establish. For my own part, I regard every portion of this doctrine as erroneous. I am glad to find in Dr. de Watteville (with whose remarks I, for the most part, cordially agree) one at least ready to hold with me that volition is only intellect in action. This doctrine I have long held; therein following Spinoza, who said more than two centuries ago: —"The Will and the Intelligence are one and the same thing." From this point of view, as I have elsewhere contended,[1] Dr. Ferrier seems to start with a fundamental misconception when he supposes, in reference to cortical centres, that those "immediately concerned in effecting volitional movements" are "as such truly motor."

The scope of "Will" or "Volition" was clearly enough marked out by Locke when he said, in the simple but effective language of the time:—"We find in ourselves a Power to begin or forbear, continue or end several Actions of our Minds and Motions of our

[1] 'Brain as an Organ of Mind,' p. 581.

Bodies, barely by a thought or preference of the Mind." As to the nature of the processes comprised under what is known as "Will," may we not find them described in the simplest terms by Hartley and his predecessor Hobbes? Thus, Hartley says:—"The Will appears to be nothing but a desire or aversion sufficiently strong to produce an action that is not automatic, primarily or secondarily. . . . The Will is, therefore, that desire or aversion which is strongest for the present time." The conflict of mental moods or motives is sometimes slight and sometimes complex (entailing what we now term inhibitory processes), before what is called a resolution or Will to do a certain action is arrived at. As Hobbes quaintly says:—"The whole sum of desires, aversions, hopes and fears, continued till the thing be either done or thought impossible, is what we call Deliberation." Here then we have intellect in action, with absolutely nothing of motor activity concerned with its manifestation.

The result of any conflict of motives, great or small, simple or complex, is that ultimately our desire to carry into action one of a certain number of plans, or to operate in this or that way, triumphs. Supposing, for the sake of argument, that the result of our deliberation is a resolve or desire to perform a certain definite muscular action; we could not resolve to perform such action without having remembered the action in question, or, in other words, without having revived in idea a conception of the movement to be executed. This was long ago clearly pointed out by James Mill, who showed that something else in reference to volitions (or actions desired to be accomplished) always accompanies or immediately follows the emotion of Desire—viz., an Idea or Conception of the kind of Movement needed for the gratification of the Desire.

Now this idea or conception of the movement needed involves the conjoint activity of two sensory centres, that is of the visual and of the kinæsthetic (under the latter would be included those tactual elements referred to by Dr. de Watteville). This idea of the movement about to be produced seems to constitute, as James Mill contended, "the last part of the mental operation," that is the last actions associated with psychic processes—beyond we have mere physical links intervening in internuncial fibres, motor centres and motor nerves, through the intermediation of which appropriate muscular contractions are caused.

Thus, the exercise of our Will means the exercise of our Intellect till, in the end, we determine upon some course of action. There is thus no separate region of the cortex needed to be set apart for volitional exercise (the idea is absurd except to those who believe

in the doctrine of Wundt and Bain) simply because all departments of mind are liable to be called into action at different times in different combinations, as the prelude to our willing this or that action—and, consequently, wide but ever-varying portions of the cerebral cortex are called into play on different occasions. Such philosophers as Dugald Stewart and Dr. Thomas Brown deliberately omitted to discuss "Will" as a distinct department of mind. "To know all our sensitive states or affections," the latter says, "all our intellectual states, all our emotions, is to know all the states or phenomena of the mind."

Suppose, for the sake of argument, that my position be correct, that the whole of the cerebral cortex is occupied with such processes as those to which I have above referred. Then it must be clear that, from this cortical mantle, fibres must descend to motor centres of various grades of complexity situated in lower portions of the brain and in the spinal cord. That being so, the question at once arises, whether these efferent fibres would pass off from all sensory centres indiscriminately. That notion I reject. I come to the conclusion, that the most important sensory guidance and education of movements has its birth either in combined auditory and kinæsthetic centres (for articulatory movements), or in combined visual and kinæsthetic centres (for limb movements).

In the case of the activity of each of these couples I come to the further conclusion, that the kinæsthetic centres are the last to come into operation. And here I am glad to have the support of Dr. Ross, who says:—"I at least give my unhesitating adhesion to Dr. Bastian's opinion, when he says that the kinæsthetic centre is the last centre which must be excited anterior to the movement in any psychical action." But the very heart or kernel of the kinæsthetic centre in any one hemisphere would, in my opinion, be represented by the cortical terminus for "muscular sense" impressions. From such regions, therefore, it is, as I suppose, that the outgoing fibres issue by means of which our Intellect plays upon subjacent motor centres when we desire to perform this or that so-called "voluntary action."

Now for one word concerning the "fiat" which Sir James Crichton-Browne seems to surround with some air of mystery, as though there at last we had a something of cortical origin which could truly be described at once as psychical and as motor. This, in my opinion, is altogether erroneous. When we hesitate as to what we shall do or say and at last decide to adopt this or that alternative, there must already during the period of hesitation be nascent excitations in the parts of the sensory centres related to the

movements thought of (that is, rival nascent ideas of movement), which would correspond with different outgoing routes for the passage of stimuli to motor centres. But as soon as we arrive at a definite desire to perform one of the movements in question, all inhibitory influences blocking that route cease (we are at the stage of what has been termed the "fiat"), the idea of this movement becomes fully revived, and the movement is straightway executed by the passage of stimuli downwards through a corresponding set of internuncial fibres issuing from the kinæsthetic centres in action, and proceeding thereby to the appropriate motor centres.

Now this conception being true, we should only expect (what is in accordance with facts) one set of excitable centres to exist in each cerebral hemisphere for the different voluntary movements an animal is capable of executing, and therefore I contend that existing evidence points strongly to the conclusion, that the centres in the Rolandic and marginal areas are, as I have termed them, kinæsthetic and not motor.

Did motor centres really exist in the cortex, clearly there should be a double set of excitable centres in each hemisphere—the one set corresponding with the definite points whence fibres pass off from the kinæsthetic to the motor centres, and the other set at the several motor centres themselves and the points whence their efferent fibres issue.

If it should be said by some, as by Dr. Ross and Mr. Horsley, that muscular sense centres may be situated in the upper layers, and corresponding motor centres in the lower layers of the same regions of the cortex, the difficulty of the absence of the two sets of centres in each hemisphere would certainly be got over. But I fully believe and hope that the persons who adopt this view may, after a time, come to see that there is really no reason for postulating the existence of motor centres in the cortex at all, when they have rejected the doctrine by which alone any such view was rendered necessary—the notion, that is, that psychical processes are direct accompaniments of the action of motor centres, and therefore that such centres would need to be correlated in the cortex with sensory centres and their derivatives. Once get rid of the notion, that any such psychical processes exist in connection with the activity of motor centres, as Dr. Ferrier and Dr. Ross have done, and there is no longer the faintest need for postulating the existence of cortical motor centres.

Critical Digest.

SOME RECENT OBSERVATIONS ON PERIPHERAL NEURITIS.

BY R. H. PIERSON, M.D. (PIRNA, DRESDEN).

SINCE the publication of Leyden's paper on Poliomyelitis and Neuritis, in 1880, which was founded on some observations made by Duménil, Eichhorst, and Leyden himself, the number of cases of peripheral (multiple) neuritis related by different authors has become so considerable that we must necessarily abstain from attempting an exhaustive review of the entire literature of the subject; we shall try, however, to give a general survey of the question as it appears in some more or less elaborate works of recent date.

To begin with English work, BUZZARD's monograph: "On some forms of paralysis from peripheral neuritis," treats the subject with all the accuracy and thoroughness that characterises this writer. The little book is divided into three lectures, the first of which contains a more general account of the various forms of neuritis, such as localised forms of neuritis (traumatic, rheumatic); typical (multiple) neuritis; alcoholic, diphtheric, syphilitic neuritis; neuritis from gout; endemic neuritis (kakké or beriberi). Dr. Buzzard justly insists upon the importance of a thorough examination of the electrical reactions in all cases where the presence of neuritis must be kept in view, and he shows in several instances the diagnostic value of this investigation. It is interesting to find here the statement, that a most precise description of typical multiple neuritis (observed in Paris) has been given forty years ago by Graves, in his "Clinical Medicine." He also states that the French pathologists "searched anxiously in the nervous centres for the cause of this strange disorder, but could find none. There was no evident lesion, functional or organic, discernible in the brain, cerebellum, or spinal marrow."[1]

[1] I have no doubt that cases of multiple neuritis might be found under different names in the literature of former years; so I came across an observation mentioned by Prof. Meynert, of Vienna, in a paper called 'Skizzen über Umfang, etc., der klinischen Psychiatrie,' p. 22 (Vienna, 1876): a waiter of most intemperate habits, having caught cold by walking on cold stones at night, complained of violent pain in the legs; is unable to walk (ataxy); paresis of the hands ensues; faradic excitability of muscles diminished; facial paralysis on the right side, dilation of left pupil, articulation impaired, total loss of sensation in both legs; death 55 days after onset of paralysis. Autopsy: Brain and spinal marrow show nothing but slight atrophy of brain and a small degree of hydrocephalus.

In the second lecture we find an accurate description of the causes, symptoms, and anatomy of multiple neuritis, more especially the alcoholic and the endemic forms of this disease. Dr. Buzzard believes that Déjerine's cases of "névro-tabes périphérique" must be considered as specimens of alcoholic neuritis, and he points out the difficulty which may occur concerning the distinction of such cases if ataxy, impaired sensibility, pains, absence of knee-phenomenon, etc., form real tabes.

The third lecture is devoted to the rarer forms of multiple neuritis, which sometimes occur after enteric fevers, dengue and malarias, and the well-known diphtheritic paralysis, which Dr. Buzzard considers to be due, in most cases, to multiple neuritis. Then follow some valuable hints about the diagnosis and prognosis of the different forms of polyneuritis, to which are added directions for the treatment of the disease.

Dr. Buzzard's book fully deserves to be studied by all those who are interested in the progress of neurology.

Of the great number of articles on neuritis published in archives and journals, we can only mention a few. ROGER [1] gives a survey of a considerable number of cases observed by several authors, adding a case of his own. This author attempts to identify Landry's acute spinal paralysis with Duchenne's paralysie ascendante subaiguë. He says that in the former the progress of the disease is too rapid to allow the development of muscular atrophy; the absence of sensitive troubles in these cases, as in Duchenne's disease, he considers as unimportant for the diagnosis, and he distinguishes therefore two forms of subacute polyneuritis according to the presence or absence of sensitive disturbances. We do not think that the present state of our knowledge justifies this statement; though since Duchenne's, Landry's, Westphal's, &c., publications on the subjects, the microscopical anatomy of the nervous centres has made considerable advances, this question may not yet be decided; from the clinical standpoint sensory symptoms are essential for the diagnosis of neuritis; and the mere fact that in some cases of Landry's paralysis no alterations of the spine and brain have been found, is not sufficient to make us look upon them as specimens of polyneuritis.[2]

[1] "Des névrites périphériques;" 'L'Encéphale,' 1885.

[2] That this view is not correct I can show by a case of Landry's paralysis which I observed in Dresden, in August 1884. The patient, an unmarried lady 47 years, had been ill about five or six days, when I was requested to see her. Dr. Hagspihl, her usual medical adviser, told me then that paralysis of both legs had been established within a few days without apparent cause. There was no fever, but great mental excitement. The patient could not move the legs at all; she could not sit without difficulty: there was not the slightest trace of muscular atrophy in the paralysed muscles; both tendon-reflexes and deep reflexes were normal; by a careful electrical examination I could not find any alteration of the faradic or galvanic reactions. Within the ensuing three or four days the upper extremities became paralysed: there was difficulty of breathing, sleeplessness, great agitation. I then ceased to attend myself; but my friend, Dr. Mossdorf, who took charge of the patient, told me that he repeatedly tested the electrical reactions without finding any change, nor was there any atrophy of

An observation published by Strümpell and Möbius[1] deserves special attention, because it proves the existence of exaggeration of the tendon-reflexes in peripheral neuritis. (Buzzard, too, has noticed this symptom in cases of alcoholic neuritis.) The authors found, in two patients, exaggerated tendon-reflexes in both arms and legs, in connection with muscular atrophy, partial R. D., and other indubitable symptoms of polyneuritis; as the patients began to recover, the tendon-reflexes became generally normal. Strümpell and Möbius are inclined to explain this phenomenon by assuming a state of increased irritability in the sensory nerves of the muscles and their neighbourhood; this is the more probable, as in both cases other sensory troubles, pain, anæsthesia, etc., were most prominent symptoms.

A great number of authors have written on the interesting form of neuritis which is not uncommonly observed in intemperate individuals. As a most accurate and elaborate essay on the subject, we have to mention Professor BERNHARDT's paper, "Ueber die Multiple Neuritis der Alkoholisten."[2] After describing a characteristic case of his own observation, Professor Bernhardt gives a detailed account of a considerable series of cases, mentioning more especially the articles of Rich, Schulz, Strümpell, Müller, and Moeli. The author then describes the recent observations of alcoholic ataxy, or "Pseudo-tabes," and their relation to tabes dorsalis; finally he treats the differential diagnosis of multiple neuritis, tabes, subacute poliomyelitis, and Landry's paralysis. When Professor Bernhardt, quoting the well-known cases of Eichhorst, Roth and Broadbent, says that we are entitled to state that after abuse of alcohol there has been observed a form of paralysis, rapidly ending by death, and likely to make the impression of Landry's paralysis even upon an experienced observer, we cannot deny this to be so; but we cannot admit that this form of paralysis has been proved to be dependent upon multiple neuritis until this will have been shown by post-mortem examination of the peripheral nerves in a case of this kind.

FRANCOTTE[3] has observed four cases of multiple neuritis, two of which ended fatally; one of them was that of a patient suffering from pulmonary consumption, in the other case there was no cause at all to be found. Microscopical examination showed in both cases

---

the muscles. The patient died suddenly three weeks after the onset of the disease, in consequence of respiratory paralysis. The autopsy was made by Dr. Birt-Hirschfeld, now professor of pathological anatomy at the University of Leipzig. There was no macroscopical alteration in the medulla spinalis. Prof. Birt-Hirschfeld intended to submit the medulla to microscopical examination, but I have not heard what the result has been. At all events, this case proves that *Roger's* suggestion, that the development of the disease is "trop rapide pour que cette alteration (muscular atrophy) ait le temps de se produire," is not acceptable. I may add that in my case there was not the *slightest sensory trouble* up to the very death of the patient, nor was there paralysis of the sphincters of the bladder and rectum, bed-sore or any of the symptoms of acute myelitis.

[1] 'Münchener Med. Wochenschrift,' 1886.

[2] 'Zeitschrift für klin. Medicin,' 1886.

[3] 'Revue de Médecine,' May 1886.

that there was degenerative atrophy of the nervous substance, more developed in the peripheral parts; the anterior roots and the spinal cord being perfectly normal.

The two other cases recovered; one of the patients was a drunkard; the other, a prostitute, had no symptoms of syphilis.

KAST, of Freiburg, has published a very interesting article in the Archiv für klin. Medicin' (vol. 40, I.). One of his cases is remarkable for its etiology; the patient, a girl 13 years of age, having had a very slight sore-throat (angina follicularis) which was followed by paresis of accommodatio , ataxy, first of the arms, afterwards of the legs, impairment of tactile sensibility, shooting pains; then ensued atrophic paralysis of the interossei and of the tongue with R. D., loss of tendon-reflexes, bulbar symptoms; death nine months after beginning of the disease; multiple degeneration of peripheral spinal and cervical nerves, brain and cord normal.

The second one was a man of intemperate habits; the symptoms of multiple neuritis were combined with swelling of several joints. The patient recovered partially. Kast considers this polyarthritis as a symptom of polyneuritis, and agrees in this respect with Strümpell. There is a considerable number of cases on record in which painful swelling of joints occurred either in the course of multiple neuritis or preceded the outbreak of the latter. Two observations of this kind have been made in 1883 by Boeck, of Christiania; one of them was combined with purpura rheumatica. Boeck suggested that this latter symptom, as well as the swelling of the joints, is due to an affection of the vasomotor nerves, caused by the same infectious agent which produces the neuritic process in the motor and sensory nerve-fibres. This explanation would be apt to account for those cases where the symptoms of acute polyarthritis seemed to appear, as it were, alternately with polyneuritis, as it was observed by Grocco and Fusari,[1] and several other authors.

We add, finally, that the statement, first made by Baelz and Scheube, that the endemic disease of beriberi or kak-ké is due to multiple peripheral neuritis, has been confirmed by Tscholowski, of St. Petersburg, who examined, post-mortem, several cases of this disease, and found well-marked degenerative atrophy of peripheral nerves together with slight atrophy of some ganglion-cells in the lumbar region of the cord, which latter alteration he is inclined to consider as a secondary occurrence.

[1] 'Annal. univers. di Med. e. Chirurg.,' 1885.

Abstracts of British and Foreign Journals.

Landouzy and Déjérine on Progressive Atrophic Myopathy involving the Face. (*Revue de Médecine*, Dec. 1886.)—The authors have devoted much time and trouble to the very careful and minute investigation of the various types which are to be found in the disease which is commonly called Progressive Muscular Atrophy (*Comptes-rendus de l'Académie des Sciences*, Paris, 7 Janvier 1884. *Revue de Méd.*, Feb.–April, 1885). They consider that the types, at least which they d[illegible] belong to a disease beginning "in" the muscle and not in the spinal cord, a conclusion they wish to indicate by calling the disease a progressive atrophic myopathy, instead of a progressive muscular atrophy. The types which they claim previously to have established are: (1) the facio-scapulo-humeral type, and (2) the scapulo-humeral; of which the second was believed to be much the more common, and the first was considered to belong to only a very few cases originating in childhood, such cases, in fact, as Duchenne had called Progressive Muscular Atrophy of Children. The chief points they wish to add to what they have said before are, that the affection of some facial muscles is much commoner than was supposed, that it consists chiefly in thickened lips, the upper generally overhanging, along with weak and unequal movements of the corners of the mouth and of the cheeks, and an incapacity to shut the eyelids completely, which gives this *facies myopathica* a heavy look and clumsy movements more easily recognised than described. The pronunciation is very indistinct, especially of the labials. And this *facies* they regard as a factor, which may come first in the symptoms of the disease either in children or adults, or may come on gradually late in the disease, or may possibly never enter into it at all. The muscular lesion which manifests itself by atrophy they consider as of the same nature as that which manifests itself by pseudo-hypertrophy; they both originate, as the most modern researches are taken to show, in a muscular irritation. That the clinical results of atrophy and pseudo-hypertrophy should be so distinct is a parallel to the cases of interstitial hepatitis, which are sometimes from beginning to end atrophic, and sometimes from

beginning to end hypertrophic. They bring forward six new cases in full detail, and in one of these was an autopsy. The first subject was a man of 36, whose grandmother, mother, and brother were atrophic, and in whom the inherited *facies myotrophica* was plain from childhood. His playmates called him a "Chinaman." He could not keep his place in a military band, because there was no chance of his ever learning to play the flute—his lips were too clumsy and hypertrophied. His scapulo-humeral symptoms on the left side were well developed at the age of 15. He was allowed to stay in the army till after the Franco-German war, when he was 21, though he only pretended to shoot from the left shoulder, and after that could do very little. He was treated in the Charité in 1885. By good fortune there were photographs of his family and himself, which showed the same type of face and atrophy in them, and the progress of the same disease. The photographs of his own case are admirably reproduced in the *Revue*. The second case was in a woman æt. 27, who came at first to the Hospital, not imagining she was ill herself, but entirely as interpreter for her paralysed mother, and in her the disease was recognised from her face only. She seemed entirely unconscious of any abnormality either in the face or the arms, but on examination the ordinary scapulo-humeral type of atrophy was found; and photographs showed that her facial symptoms had existed from childhood. She could not whistle, or blow out a candle, or frown, or shut her eyes completely. Her lips were thick, and moved very little in speech or laughter; there was reaction of degeneration in the *orbicularis oris*.

The third subject was a man, born in 1856, in whom some affection of the face was noticed when he was three years old. Some atrophy of the muscles of the trunk and legs came on two years later, and very gradually spread to the arms. When he was nine he was taken to see Duchenne at Boulogne, who was very much interested in the boy, as illustrating that rare form of disease which he had then (1868) named atrophic paralysis of children. He published an account of the case (*De l'Electrisation localisée*, 3me éd. 1872, p. 1098), fully describing the facial symptoms mentioned in the other cases, except the inability to close the eyes completely; he illustrated them by photographs, and found similar symptoms in his mother and his brother. A year or two later the flexors of both legs began to contract, and he became a hopeless cripple. He could use his hands, however, and made a scanty living by being dragged about France in a go-cart by an idiot, and selling little

books of instruction in shorthand writing. At last the idiot died and he came into Hospital under the care of M. Landouzy in 1886, showing the same *facies myopathica*, of which Duchenne had published a description 18 years before, with the additional inability to close the eyes completely which had not been noticed previously. The atrophy in the arms and neck were much more advanced. In the 4th case there was no history of heredity; atrophy of a scapulo-humeral type came on æt. 40; and no affection of the face was noticed till he was 45. In the 5th case there was probably some complication with lead-poisoning. He was a man of 40, who had had dropped wrist in 1872, and in consequence given up his trade as painter; but in 1885 was found to have progressive atrophy of scapulo-humeral type, with the addition of facial symptoms in 1886 and notable atrophy of the tongue. The 6th and last case, in a man of 66, was fatal, and was examined very minutely. At the age of 20 he began to notice atrophy about the shoulder, which spread very slowly into the arms and legs. He was a hawker, and could go on with his trade till he was 63. There was no visible affection of the facial muscles whatever; no fibrillar contractions; no reaction of degeneration; no abnormality of knee-jerk and no hereditary history. He died of pulmonary-tuberculosis, and an autopsy showed no abnormality of the spinal cord, of the cervical sympathetic, of the anterior roots of the peripheral nerves; but simple atrophy of the muscular fibres with great multiplication of the nuclei. In some muscles a few fibres were hypertrophied, and in the most atrophied muscles there was some interstitial fat. The point on which the authors lay most stress is that, though they could not detect abnormality of the facial muscles in life, they found after death that they were degenerate or beginning to degenerate, a point they consider of importance as showing that, sooner or later, the face becomes involved in this progressive atrophy. Westphal has also published three cases of progressive muscular atrophy with affection of the ocular muscles (*Charité-Annalen*, Berlin, 1886), and he notices the thick, overhanging upper lip, "like a tapir's," in one of them.

A. T. Myers, M.D.

BRAIN.

JULY, 1887.

Original Articles.

THE NATURE OF THE OBJECTIVE CAUSE OF SENSATION.—PART II. TASTE.

BY JOHN BERRY HAYCRAFT.

HOBBES (subsequently Hartley and many English psychologists) viewed sensation as the result of a pressure or action of an external particle of matter upon the appropriate sense-organ, and a transmission along a nerve to the brain. Those multitudinous feelings of sight and hearing, of taste and smell, of which we are conscious, were all, according to this thinker, the result of transmissions to the brain excited from without; nor is this view without modern scientific support.

It is not one of the newest triumphs of microscopical research to show that the eye, the ear, the nose, and the tongue are all beset with little cellular particles, attached to the sensory nerves of those parts, and extremely sensitive to the "pressures" or "actions" of external particles of matter. Let us call these the sensitive cells or end-organs of the body, understanding by this term that they are formed on the external ends of the nerves. These end-organs are all very much alike in shape and general appearance, it matters not from which sense-organ you take them for examination. In all cases the part attached to the nerve is thick, granular like ground glass, and with a kernel or nucleus; the outer and more projecting part is drawn out into a rod or hair. It is the

style or hair which, directly stimulated from without the body, excites the nerve through the inner segment, sending an impulse to the brain, and giving rise to a sensation.

No doubt an anatomist could, by careful inspection, tell at once the end-organs, taken from the retina of the eye, from those of the ear, or the nose, yet they are all formed, as it were, on the same plan.

No one has insisted more emphatically than Mr. Herbert Spencer that these end-organs have all come from common parentage, hence, in part, their similarity. We are to look, in fact, if we wish to understand their nature aright, upon these different end-organs as arising, by slow development, during the evolution of the animal world, from a layer of simple cells—ectodermic—found covering the bodies of our more primitive ancestors. Most of these ectodermic cells remain as the cells of the skin, but some are changed into the end-organs of the special senses we are discussing.

Now this development, Mr. Herbert Spencer would say, is in part the result of the action of the surrounding forces or the environment upon the organism itself. These acting for many generations have produced changes which depend upon the nature of the organism and of its environment.

That motion of the ether which travels with such velocity and which, striking the eye, sets up an impulse in the optic nerve, and on reaching the brain produces a sensation we call light, is a vibratory motion.

That motion of the air—not of the same velocity as light—which, striking the ear, produces a sensation we call sound, when the impulse it sets up reaches the brain along the auditory nerve, is also a *vibration*.

No one doubts at the present time that all material particles are in constant motion of a vibratory nature. The molecules of a gas are vibrating; so are those of a liquid and a solid. The particles of salt in the sea, or the sugar in a cup of tea, are in constant motion; so are the molecules of quinine and quassia, the odorous particles of musk and of the rose.

We see then that the body is surrounded and acted upon by various forms of matter in vibratory motion. The environments which have developed the special organs out of

the more simple ectodermic cells are moving particles of matter, and they have in all the sense-organs produced the end-organs before alluded to—armed with little rods or styles, the identical structures capable of being to an extraordinary degree affected by motion.

With these general qualities common to the environments of all the special senses, which senses are built up so essentially on the same type, we shall expect to find very striking analogies between the modes of production of the different senses. Thanks to the labours of Young, Maxwell and Helmholtz, we know a good deal about the way in which the organism is stimulated by light and sound, but we know almost nothing about the production of taste and smell. Indeed this matter has as yet received so little attention that scarcely anything is known to the specialist which is not the intellectual property of every one.

The following paper is an investigation into the nature of the production of taste sensations; the question of the production of smell sensations I shall discuss in a subsequent paper.

Let us take for our guide this unity of plan so evident from a study of the microscopical anatomy of the sense-organs; let us study, first the production of sight and hearing, and then endeavour to find analogous ways in which it may be possible to account for the production of taste.

The end-organs of the retina are stimulated by the vibrations of that rarefied matter called ether, in a way as yet not clearly understood. These vibrations differ very much from one another in rapidity or pitch, some are so slow—the ultra-red rays of the spectrum—that they produce no sensation at all. Some again —the ultra-violet—produce no sensation, because they are too rapid. Between these two extremes we find a large number of rays of the visible spectrum—which when they fall upon the eye cause a sensation of light and colour. Now it is found that the kind of colour, or, as we may call it, the quality of the sensation, depends directly upon the pitch of the vibration and nothing else. A somewhat slow vibration causes a sensation of red, a more rapid one a sensation of orange, and so on. It is seldom in nature that the eye is stimulated by vibrations of

only one pitch; thus I have in front of me a book that has a cover of a crimson colour, and I know by experiment that this crimson sensation is only produced when the eye receives at the same time from the same object at least two sets of vibrations. The vibrations that produce the sensations of crimson, scarlet, purple, lilac, &c., are all complex; that is, they are combinations of two or more sets of simple vibrations.

We see then that it is the *kind of vibration* which determines the quality of the sensation in the sense of sight, and that there are differences in the vibratory ether outside the body to correspond with the almost limitless colour variations of which we are sensible.

Let us turn to hearing. If we place a long piece of steel in a vice, and alter its length at will, we shall find that on striking it with a rod it will vary in the rapidity of its vibrations, and various sound sensations will be produced.

If it vibrate slowly, some ten times a second, we shall hear nothing, and if it vibrate very rapidly indeed we shall hear nothing; but, as in the case of sight, we shall find varying sound sensations produced within these extreme limits. The quicker the vibration, the shriller the sound.

In nature, observe the analogy with the ether vibration; sonorous vibrations are seldom simple. From a flute, or a violin, we have a bundle of vibrations striking the ear when a note is sounded. The vibration is complex—generally consisting of a slow vibration called a fundamental tone, and a series of overtones harmonically related to it. The sound of a flute is quite different from that of a violin, even when the same note in the musical scale is sounded in both cases. The reason is that the bundles of vibrations are different, the overtones differing in intensity and arrangement. We see therefore that the *kind of vibration* falling on the ear determines the quality of the sound produced. Both in sight and hearing the sensations will depend upon the pitch or rapidity of the vibration if it is a simple one; or if it is complex, upon the pitch and arrangement of the component simple vibrations. We may say then that the kind or character of the vibratory motion determines the quality of the sensation in both cases. A knowledge of these facts, together with the known similarity

in structure of the end-organs of all the senses, will naturally lead an investigator who wishes to study taste or smell production to search for analogous conditions in these senses also.

In sound and light we have motions varying to an almost limitless degree in their kind or character. Have we motions of the same or similar nature in rapid solution?

As we have seen, matter in the liquid state is in motion, the molecules of sugar in water are in motion, and what is more, the motion is characteristic of the substance. The motion of salt is different from the motion of sugar, and the motion of sugar from the motion of quinine. We have then a field for inquiry open to us, for if we can connect definite taste sensations with definite motions we shall have the analogy we seek. Let us proceed to this inquiry. We know that by increasing the weight of a tuning-fork, and also by increasing its bulk, we can cause it to vibrate more slowly, and the same law has been found to hold good for ultimate atoms and molecules, the proof of which will be given later. Is there then any relation between taste sensations and the molecular height of sapid substances?

If we take the molecular weights of acid substances, we shall find in the case of hydrochloric acid a weight of 36·5, in the case of sulphuric acid 98. Lithium chloride, molecular weight 42·5, and potassium sulphate, molecular weight 174, are saline. Many other examples might be adduced from bitter and sweet compounds, to demonstrate that we cannot tell by the molecular weight alone anything about the taste of a substance. In the above example, an acid may have a lower or a higher molecular weight than a salt.

We shall see hereafter that molecular weight is not without its importance, and even at this stage of our inquiry it may be well to allude to a series of facts in this relationship. Bodies which have the greatest molecular weight of any, such as albumens, albuminoids, starches, gums, &c., are totally without taste; nor need it be urged that a ready explanation of this is at hand. No doubt many of these substances are not readily soluble in water, and in consequence they will not readily permeate the viscous layer of saliva covering the tongue and the surface of the gustatory end-organs. Some

albumens, such as serum and egg albumen, are fairly soluble, and they may all be converted by appropriate ferments into allied products called peptones, which are soluble and readily diffusible, and nevertheless devoid of taste. In like manner, starch may be converted into dextrin, a substance readily diffusible in water, and tasteless.

We find also among the list of tasteless bodies all those of very small molecular weight. Water, molecular weight 18, is an example. The simple gases dissolved in water are tasteless, but when we examine substance with a molecular weight over 30, we find that when dissolved in water it is capable of producing some taste or other. Sulphurated hydrogen, molecular weight 34, is very slightly acid to the taste; hydrochloric acid, molecular weight 36·5, is very acid; and nitrogen monoxide, molecular weight 46, is sweet. It is true that the monatomic alcohols, such as ordinary commercial alcohol, have molecular weights over 34, and are tasteless, so that the same rule does not apply to all the carbon compounds; but even here we are struck with the fact, that these alcohols are the lowest of a series, all the polyatomic and more complex alcohols being sapid and sweet in character.

There seems then to be some analogy between taste on the one hand, and sight on the other. The substances of lowest molecular weight, and therefore of most rapid vibration, like the ultra-violet rays of the spectrum, are incapable of producing sensation. In like manner, there are substances of very great molecular weight, and of the most sluggish molecular vibration like the vibrations of the ultra-red, also incapable of affecting the sensorium.

Let us turn again to our study of sapid substances, restricting ourselves in the first case to the investigation of inorganic compounds. A large number, perhaps the greater number, of inorganic compounds are insoluble in water, and are therefore tasteless; we are therefore confined to those which are soluble, and which are chiefly the salts of certain metals. I have endeavoured to obtain information as to the tastes of different substances, by consulting standard works on chemistry, especially the dictionary edited by Watts. In many cases I was unable to obtain any information at all, and in nearly

every case it was insufficient for my purpose. One knows pretty exactly what is meant by a peacock blue, or a salmon colour, but the general descriptions of tastes are absolutely unreliable. Common salt and nitre are both described as saline, sulphate of magnesia and quinine both as bitter. I have been obliged on this account to taste the different substances for myself, and all the observations recorded in this paper are the result of at least two careful experiments with each substance. By the term *salt* I shall indicate a taste like that of common table salt; by *saline*, a taste like that of nitre.

In recent times a remarkable discovery of Newlands has formed a fresh point of departure in connection with the science of chemical physics. His observations led him to formulate a law which he termed the law of octaves. Lothar Meyer, Mendelejeff and Carnelley, in extending his work, have shown that the Periodic Law—as it is now termed—is one of wide application and importance. The nature of the Periodic Law is now so well known, thanks to the many recent publications of Professor Carnelley, that it would be superfluous to do more than merely sketch its main features.

If we arrange the elements in the order of their atomic weights, beginning with that which has the lowest, and passing to that which has the highest, we shall find that there is a periodic recurrence of function or property in the series. The first element is a monad, the second a dyad, the third a tryad, and the fourth a tetrad. Then we find the fifth a triad, the sixth a dyad, and the seventh once more a monad. Then follows a second series of seven elements, showing the same variation in atomicity, and this repeats itself right through the list of elements.

This periodic recurrence is seen not only with atomicity, but with the atomic volume, the fusibility, the electrical and other properties of the elements. There is then a general resemblance in physical properties between the first, eighth, fifteenth, &c.; between the second, ninth, sixteenth, &c., and so on. Mendelejeff has arranged the elements in a very convenient tabular form, see table p. 152, which brings out these and some other important facts.

Those elements which resemble one another, and which we

can pick out, taking every eighth one from that one we start with, form what he calls *a group* (see table). In vertical series are arranged the *groups* of similar elements, and the *series*, each of seven elements, are seen on the horizontal line.

TABLE OF NATURAL CLASSIFICATION OF ELEMENTS. AFTER MENDELEJEFF.

Group	I.	II.	III.	IV.	V.	VI.	VII.	Group VIII.
Series.	Monads.	Dyads.	Triads.	Tetrads.	Triads or Pentads.	Dyads or Hexads.	Monads or Heptads.	
1	H=1	..	..	..	..	..	..	..
2	li=7	be=9	b=11	c=12	n=14	o=16	f=19	..
3	NA=23	Mg=24	AL=27	SI=28	P=31	S=32	CL=35·5	..
4	K=39	ca=40	sc=44	ti=48	v=51	cr=53	mn=55	fe=56 co=57 ni=59
5	CU=63	ZN=65	GA=69	..	AS=75	SE=79	BR=80	..
6	rb=85	sr=87	y=89	zn=90	nb=94	mo=96	..	ru=104 rh=104 pd=106
7	AG=108	CD=112	IN=113	SN=118	SB=120	TE=125	I=127	..
8	cs=133	ba=137	la=139	Ce=142	di=147	..	..	..
9	..	..	..	..	ER=166	..	..	..
10	..	..	yb=173	..	ta=182	w=184	..	os=193 ia=193 pt=195
11	AU=197	HG=200	TI=204	PB=207	BI=210	..	..	..
12	..	..	..	th=234	..	u=240	..	..

There is one other point of importance, namely, that the elements of a *group* which are in an *even* series are especially related one to another in their properties. In the table they are represented in small type. So, in like manner, elements belonging to the same group of *odd* series are especially related.

Thus Li, Na, K, Cu, Rb, Ag, Cs, Au have all of them properties in common; but in this group Na, Cu, Ag, Au are most alike, and Li, K, Rb, Cs, also in like manner, are especially related.

Scientific men have long been taught to expect that material substances similar in their ordinary physical properties, will exert a similar influence on organic life. No one has more completely demonstrated this than Drs. Lauder Brunton, Fraser, and Crum Brown, who have shown that not only elementary protoplasm, but even highly differentiated muscular and nervous tissues are affected in a similar way by substances having allied chemical and physical properties.

Every one is aware, too, of the important part played by the component atoms of a molecule, how by replacing an atom of

hydrogen by an atom of potassium one can convert ordinary water into a caustic alkaline substance. Inasmuch as we have a number of salts formed, say, by the union of chlorine with another element, it is a question well worth the discussion, whether salts formed by the union of chlorine with elements of the *same group* have similar tastes. Speaking more generally: do elements belonging to the same group when combined with the same element or group of elements to form salts, similar as they are in physical properties, produce similar tastes?

It was this question which occurred to me, when, baffled in the hope of connecting taste directly with the gross atomic weight of the sapid substance, I had to reconsider the question afresh. This question, I shall hope to show, may be answered in the affirmative.

By far the greater number of sapid substances contain elements found in Groups I., II. and VII., and these I accordingly studied. The salts of Groups I. and II. most easily obtained are the chlorides and sulphates, and of Group VII. I selected the combinations these elements form with sodium and potassium. As it was very necessary to use salts as pure as possible, many were prepared for me with great care by Mr. Harris the chemist, but for the greater number I have to thank my colleague Dr. Nicol, who placed at my disposal a large collection of very pure salts prepared by himself for his own well-known investigation on solubilities.

In the subjoined tables are the results of an investigation of Groups I., II. and VII. We have then to see whether the same taste sensations are produced by the chlorides, &c., of elements having similar physical properties.

GROUP I.

Metal.	Chloride.	Sulphate.
L	Salt	Sal. Bit.
Na	Salt	Sal. Bit.
K	Salt Sal. *Bit.*[1]	Sal. Bit.
Cu	Insoluble	Sal. Bit.
Rb	Salt *Bit.* Sal.	..
Ag	Insoluble	Sal. Bit.
Cs	Salt *Bit.* Sal.	..
Au	Sal. Ast.	..

[1] *Italics* indicate that the sensation is but slightly perceived.

It will be seen that all the soluble chlorides are salt like table salt, although with the higher members we have the taste becoming more saline, a very slight bitter taste also develops. Some salts are astringent, as will be seen in a study of this and of other groups. I do not think this astringency is generally looked upon as a taste at all; depending, as it does, upon the formation of insoluble albuminates, it is purely a local action on the mucous membrane of the tongue, and will not be again discussed.

The sulphates are saline, not salt at all, and in addition they are distinctly bitter. I have not been able to obtain the sulphates of cæsium and rubidium.

GROUP II.

Metal.	Chloride.	Sulphate.
Be	..	Acid. Sweet. Ast.
Mg	Bit. Sal. Wrm. Pung.	Bit. Sal.
Ca	Bit. Sal. Wrm. Pung.	Insoluble
Zn	Bit. Sal. Wrm. Pung.	Bit. Sal. Ast.
Sr	Bit. Sal. Wrm. Pung.	Insoluble
Cd	Bit. Sal. Wrm. Ast.	Bit. Wrm. Ast.
Ba	Bit. Sal. Pung.	Insoluble
Hg	..	..

In Group II. the chlorides are all bitter salines with, in nearly every case, a warm pungent flavour. They are very disagreeable to taste, and it is long before the flavour leaves the mouth. Beryllium chloride with the sulphate is described as sweet. I have not tasted the chloride; the sulphate is distinctly acid and astringent as well. Beryllium salts then are a well-marked exception to the other salts, giving, as they do, such constant taste sensations. The atomic weight of beryllium was long an open question. I am informed on competent authority that there is now no doubt on this head, and I am at a loss therefore to explain its action as a sapid substance. The soluble sulphates are saline and bitter.

In Group VII. we have tastes produced which are in the main salt. In the case of both the sodium and potassium compounds there is a tendency to the production of the saline and bitter among the higher members of the group, and this is sooner seen in the case of potassium—itself a higher

GROUP VII.

Element.	Sodium Comp.	Potassium Comp.
F	Salt	Salt. *Sal.*
Cl	Salt	Salt. Sal. *Bit.*
Mn	..	..
Br	Salt. *Sal.*	Salt. Sal. *Bit.*
I	Sal. Bit.	Sal. Bit.

member—than in the case of sodium, a lower member of Group I.

From a study of these groups we may learn many important facts. It will be seen that much depends upon the electro-negative group with which the element is combined. Thus sodium combined as a chloride has a different taste from the sulphate of the same element: with the same electro-negative group similar elements give similar tastes, but with a curious and very uniform change as we pass from an element of low to one of high atomic weight. As we shall soon see, a change in physical property may, in like manner, be seen as we pass from a lower to a higher member of a group.

So far then we have reduced taste to a *function* of elements and their compounds, and we see that it obeys laws which are the same for so-called physical properties of these elements. Just as from a knowledge of the components of a compound we can account for its physical properties, so we have to take to pieces the sapid substance, before we get the clue to relationship between its nature as indicated by its chemical and physical properties, and the sensation it produces.

This is much already to learn; but can we go a step further and ask "in what *essential* are elements alike that produce similar tastes?" We turn naturally to the question with which we started, and ask, "do these elements vibrate in any way that is similar?" Rapid advances are being made in the more exact and extended inquiring, how do the ultimate particles of matter move? In a few years, no doubt, the investigations into the ultra-red and ultra-violet spectrum will shed a flood of light on this question, and will enable us to come to some more definite conclusion than at present. Still much is known, and it may not be premature even now to make use of this incomplete knowledge.

No one would expect to find very closely allied spectra when comparing elements even of the same groups, and for this reason. Suppose potassium and lithium to have such a fundamental tone and a series of upper partials each in an exactly similar inharmonic ascending scale, the tones of the heavier—potassium—would all be of lower pitch than the lighter—lithium. An absorption line of potassium in the visible spectrum will correspond in this case to one of lithium in the ultra-violet. Thus all attempts to establish exact mathematical relationships between the wave lengths of similar metals are for the present out of the question. We can but hope to find rough points of similarity, and these are forthcoming. The chlorides of the alkaline earths are nearly related. The lithium and sodium spectra have some points of similarity, especially in their extreme simplicity; also potassium and rubidium, each with their five groups of lines. Then again, chlorides, bromides, and iodides of calcium and barium are nearly related, the lines shifting towards the red end of the spectrum in a way which is nearly proportional with the increase of atomic weight.

Many salts absorb light of different wave lengths, and are in consequence of a definite colour. The colour is an index to this absorption, and to the state of molecular vibration of the salt molecules. In a paper on the colour of chemical compounds, recently published in the philosophical magazine ('Phil. Mag.,' July 1884), Professor Carnelley demonstrated a relationship between salts of metals of the same group in respect to colour. The salts, say, the chlorides, of a group of metals, are much of the same colour, except that, as you pass to the higher members of a group, this changes somewhat, shifting uniformly towards the red end of the spectrum. This is illustrated by the following diagrams taken from his paper, in which only those metals of a group are given, which are in alternate series.

Metal.	Cl.	I.	Metal.	CrO_4.	AsO_4.
Na	White	White	Mg	Lemon Yellow	White
C	White	Cream	Zn	Yellow	White
Ag	White	Light Yellow	Cd	Orange Yellow	White
Au	Yellow White	Golden Yellow	Hg	Red	Yellow

The striking analogy between the above tables and those of taste already alluded to, needs scarcely to be pointed out, it is obvious.

But what does this shifting towards the red end of the spectrum indicate in the case of these coloured salts?

Probably the colourless ones have vibrations of high pitch in the ultra-violet; with a higher atomic weight and slower vibration they gradually absorb the rays of the visible spectrum. The blue rays are first absorbed, and the salt appears yellow; then the green giving orange, and then the yellow and orange rays are also absorbed giving red.

In the case of tellurium chloride, all are absorbed, and the salt is black. Carnelley has therefore demonstrated that the salts of a group have molecular vibrations which are similar, which absorb light and give rise to *colour sensations* which are similar, changing uniformly, however, with increasing atomic weight.

Here then is the whole analogy between taste and sight, and it is most complete in its nature. If a curve be constructed in which the ordinates represent the atomic weights of the positive elements, and the abscissæ a chromatic scale rising from blue, green, &c., to black, we shall obtain a curve indicating that the colours of the compounds are a periodic function of the elements arranged in atomic series. This is best seen in the case of the normal iodides.

Upon the pitch of the vibration depends the colour sensation, as every one would admit: we find in the case of taste a result in every way is the same.

This has been one induction from the study of the inorganic compounds, but a whole field of inquiry had been untouched. As yet no allusion has been made to the carbon compounds, many of which are sapid. One of the most important and interesting facts in connection with the chemistry of the carbon compounds is, that they have what may be termed a *structure*. Thus the formula for common alcohol is C^2H^6O, but one of the hydrogen atoms is found to be especially related to the oxygen, and it is written C^2H^5 (OH). In tbis case then there is one group (compound radical) C^2H^5 united with another group OH. Now these

groups play the same part that elements do in inorganic compounds, and it is necessary to know this so-called structural formula in order to understand the property of an organic substance. Just as one can replace the hydrogen in water with potassium, and form another substance of different properties, depending upon the nature of the substance added, so, in like manner, one can replace the C^2H^5, or the OH, by other compound radicals, or even elements, and produce a substance whose properties will depend upon the nature of the radical you add. There are then two points for investigation. In the first case, can we find in sapid substances, having, say a sweet or a sour taste, any compound radicals always present, and from whose presence we may say that the taste sensations result? The chemist associates certain properties with the presence of a certain radical in a substance; can we find similar associations with taste sensation?

In the second place, do we find with the spectroscope that the various combinations of a compound radical preserve the character of the vibrations of the radicals, unaltered in any marked degree?

The answer to the first question can be readily given, for it will be sufficient to obtain a list of substances giving a definite taste, and to search for some common or similar radical. If a radical common to all be found, we may safely answer it in the affirmative. There are among the carbon compounds many substances having an acid taste. Here is a list of those most familiar:

Acetic acid .. .. $CH_3\,CO\,.\,OH$

$$\begin{array}{l}CO\,.\,OH\\ |\\ CO\,.\,OH\end{array}$$
Oxalic acid .. ..

$$\begin{array}{l}CH_2\,.\,CO\,.\,OH\\ |\\ CH_2\,.\,CO\,.\,OH\end{array}$$
Succinic acid .. ..

$$CH_2\begin{cases}OH\\ CO\,.\,OH\end{cases}$$
Glycolic acid .. ..

$$\begin{array}{l}CO\,.\,OH\\ |\\ CH\ \ OH\\ |\\ CH_2\,.\,OH\end{array}$$
Glyceric acid .. ..

$$CH_3\,CH\begin{cases}OH\\ CO\,.\,OH\end{cases}$$
Lactic acid .. ..

Tartaric acid	$CH(OH)CO.OH$ $\vert$ $CH(OH)CO.OH$
Benzoic acid	$C_6H_5.CO.OH$
Malic acid	$CH(OH).CO.OH$ $\vert$ $CH_2.CO.OH$
Glyoxylic acid	CHO $\vert$ $CO.OH$

The above ten examples of substances possessing distinct acid tastes are taken from various classes of organic acids, and they will be seen to have in all cases a common group of elements. This group is a compound radical CO . OH, which is combined in all cases with a different molecule. We have then the same right to impute to this radical the capacity of producing a given taste sensation, as the chemist has of looking upon it as related to certain physical properties of the compounds in which it may be found.

Among sweet substances we find the following:

Glycol..	$CH_2.OH$ $\vert$ $CH_2.OH$
Glycerine	$CH_2.OH$ $\vert$ $CH.OH$ $\vert$ $CH_2.OH$
Mannite	$C_6H_8(OH)_6$
Glucose	$C_6H_{12}O_6$
Inosit	$C_6H_{12}O_6$
Saccharine	$C_{12}H_{22}O_{11}$

These are all alcoholic bodies, glycol, glycerine, mannite being, respectively, diatomic, triatomic and hexatomic alcohol. The rational formulæ of the last three and other nearly related substances, such as levulose, maltose, &c., are not at present definitely settled; but it is certain that they contain a radical CH_2OH, found in the first three substances. Thus the formula of glucose, according to Colley, is—

$$CH_2.OH - CH.OH - CH.OH - CH.OH - CHOH - .CHO$$

Monatomic ethyl alcohol is tasteless, and has been alluded to already. It is the simplest alcohol, the polyatomic alcohols referred to having a sweet taste.

We see here again that, with a definite molecular structure, with the presence of certain groups of elements, a definite taste sensation is produced. A chemist examining the rational formula of a substance will predict its properties to an extent which will vary with the extent of his knowledge. We too can predict a property, that of producing particular sensation when applied to the tongue.

There is a large number of organic compounds having a bitter taste, such as quinine, quassia, strychnia, &c.; but inasmuch as so little is known as to the chemical nature of these bodies, an investigation, if applied to this class of substances, would be of little use.

We have seen that in inorganic compounds the physical, chemical, and taste properties vary with the nature of the elements in the sapid substance; with similar elements similar properties are associated. So too we have seen among the carbon compounds that the physical, chemical, and taste properties vary with the compound radicals present in the sapid substance. The question now to be determined is, whether or not the compound radicals behave as elements, vibrating in a characteristic and definite way, not materially disturbed by altering the combination? This may be determined by the eye, with or without the aid of the spectroscope.

We know that chromic acid has an orange-red colour; combine it with metallic elements to form chromates, and we find that these are coloured, the colour varying very slightly with the metal.

Picric acid is a coloured solid, and it probably owes its colour, at any rate in part, to the presence of a radical (NO_2). Its salts too are coloured, the absorption shifting towards the red end of the spectrum.

In his interesting researches into the spectra of colourless fluids, observed through a considerable length of tube, Dr. W. S. Russell finds that ammonia gives certain well-marked absorption bands. If an atom of hydrogen in the ammonia be replaced by methyl, forming a substance methylamine, the ammonia bands are still seen, shifted however slightly towards the red end of the spectrum.

$$\begin{array}{c} H \\ | \\ N - H \\ | \\ H \end{array} \qquad\qquad \begin{array}{ccc} H & & H \\ | & & | \\ N & - & C - H \\ | & & | \\ H & & H \end{array}$$

Ammonia   Methylamine.

Replace the hydrogen by the higher group C_2H_5, or ethyl, to form ethylamine, and the shifting continues towards the red.

With a series of alcohols, such as methylic, ethylic, propylic alcohol, we find, as we pass to the more complex, heavier, but similarly constituted molecules, a shifting of equally characteristic bands towards the red end of the spectrum. It is very much (and here we may recall the colour experiments of Professor Carnelley) as if we took an instrument, say a violin, and kept loading its strings. The notes would be heard deeper and deeper in tone, but it would be still recognised as being the tone or quality of the violin.

I have mentioned the above instances, though many others might have been adduced to demonstrate the same fact. We have every reason then to believe that the radical $\begin{array}{c} -C-O-H \\ \| \\ O \end{array}$ and the radical $\begin{array}{c} H \\ | \\ -C-O-H \\ | \\ H \end{array}$ of the acid and sweet substances are both in constant and characteristic vibration, although this vibration does not effect the rays of the visible spectrum. Not only so, but we know that this vibration will not be altered in character (from the analogy with the ammonia radical in methylamine), being merely shifted to a small extent in pitch, when its combinations are changed. This is, however, the analogy which we have sought.

Both in the case of sight, hearing, taste, and, as I hope afterwards to prove, in the case of smell we have these analogous phenomena. On the side of sensation we have variations in what we may call quality; on the objective side we have matter in vibration—these vibrations vary in character; and it is possible to connect the quality of the sensation with the character of the vibration.

It may be urged that inasmuch as an acid like hydrochloric acid produces much the same taste as oxalic acid, there

is no such definite connection between vibration and sensation. There are, too, some sweet substances having no relationship to an alcohol. We know that one can get the same colour sensation from a solution of either a picrate or a chromate, two substances absolutely dissimilar in chemical properties. The reason is that they both give a group of vibrations in the same part of the solar spectrum. It would be reasonable to expect that instances would be forthcoming of substances of dissimilar nature and chemical composition, with similar tastes. It would seem probable then that there is a scale of vibrations, which may stimulate the taste organs analogous to the visible spectrum. If a substance vibrates in a definite part of this scale, a definite taste will be produced.

One must always remember that the tongue has, like the eye and unlike the ear, no power of analysis. It may be that just as a colour, say orange, may be produced by either a simple vibration, or a combination of vibrations, some higher and some lower in pitch than that of the simple one, so the same taste may, in like manner, be produced by a simple vibration or a compound one made up of simple vibrations of higher or lower pitch.

In conclusion, it will be necessary to state very emphatically that it is *analogy* that this paper proves—analogy in the production of the senses. It would be premature to say definitely how the vibrations of common salt produce irritation of the gustatory nerve. Is it by setting up a sympathetic vibration, when, as in the ear, the sonorous waves set in motion the end-organs of the labyrinth? Upon such a question I have no wish to hazard what could only be an opinion? In the eye, vibrations stimulate the cones. How is this done? It is quite conceivable that this is by setting up sympathetic vibrations, but many suppose it to be a chemical action.

If we ask what is a chemical action, no adequate reply will be forthcoming, though many would hesitatingly affirm that even so-called chemical actions may have a vibratory and mechanical interpretation. When we say that we *understand* the manner in which the end-organs of the ear are stimulated, we mean that we can prove an analogy between the sympathetic vibration of a tuning-fork and of the organ of Corti,

when affected by the same wave motion. It is nothing but an analogy after all.

The production, on the other hand, of end-organ irritation, by light or molecular vibration, has no sufficient definite analogue in the laboratory of the experimenter.

All that I claim to have proved is, that the body is surrounded by vibrating of matter, stimulating its sensitive surface; that in all cases the process of stimulation with the production of a consequent sensation is the same to this extent; that the quality of sensation is dependent upon the character (pitch and complexity) of the vibratory matter.

Just as a certain class of salts of allied chemical and physical properties vibrate in a similar way, and stimulating the eye, produce the same colour sensations; just as certain strings of definite length and consistency vibrate in a similar way, and produce the same sound sensation—so, in like manner, similar sapid compounds (containing similar elements, or the same compound radicals) vibrate in a similar way, and produce the same taste.

ON RECURRENT PALPITATION OF EXTREME RAPIDITY IN PERSONS OTHERWISE APPARENTLY HEALTHY.

BY JOHN S. BRISTOWE, M.D., LL.D., F.R.S.

THE subject to which I wish to direct attention is that of extremely rapid pulsation, occurring for the most part in intermittent paroxysms of variable duration, in hearts structurally and texturally sound, and in persons otherwise healthy.

That hearts may beat with the extreme rapidity with which I have found them to beat, is a fact which, I think, has been largely overlooked, and with which I, at any rate, had no practical acquaintance until within the last two or three years; and yet I feel sure, judging from my recent experience, that the condition which I am about to discuss is of frequent occurrence, and needs only to be looked for intelligently to be recognised in many persons who are regarded as merely nervous and liable to attacks of ordinary palpitation.

So far as I know, the literature of the subject was, until recently, limited to the report in the 'British Medical Journal,' for the year 1866, of three well-marked cases, the first from the pen of the late Dr. Cotton, and the others respectively by Dr. James Edmunds and the late Sir Thomas Watson. Of these cases I need only say, that they almost accurately resembled the most striking and typical of the cases which are incorporated in this paper; that the cardiac pulsations during the paroxysms of palpitation were at the rate of about 240 in the minute; that in the intervals the patients appeared to be well and free from heart disease; and that in Sir Thomas Watson's case (which proved suddenly fatal) the heart was found to be soft and somewhat enlarged, but otherwise healthy.

The first typical case of the disease which I ever fully recognised was one which I saw in consultation with Dr. Wyman, of Putney, in the early part of 1885. The patient was a fairly healthy-looking young married lady, who had evidently been liable for some years to attacks of palpitation, and was free from structural disease of the heart. The attack in which I saw her came on suddenly, without apparent cause, and after a week left her as suddenly as it had arisen. Her pulse varied between 180 and 192 in the minute. A few weeks later she had a recurrence of palpitation, when the cardiac beats were counted at 246. What seemed to me at the time the most remarkable feature of her case was the apparent absence of distress. Had I not known that the patient's heart was beating with extraordinary rapidity it would never have struck me, from watching her and conversing with her, that there was anything the matter with her.

CASE I.—*Paroxysmal Hurry of Heart of Some Years' Duration.*

Mrs. P., a married lady 30 years of age, was attacked about six weeks before I saw her with pleuro-pneumonia of the right side, from which she had apparently recovered completely at the end of about four weeks. A few days before my visit (April 5th) she had taken a drive. This seems to have upset her; for on her return home she was attacked with retching, dyspnœa and nervousness. The retching soon ceased; but the pulse was found to be beating with great rapidity (about 180 in the minute), and this rapid pulsation has continued. She has been kept in bed, complaining of fluttering in the region of the heart, slight dyspnœa and nervousness; but otherwise has appeared fairly well. When I saw her she was sitting up in bed, looking bright and cheerful, but pale and delicate. There was no obvious dyspnœa, although perhaps she breathed a little more quickly than normal, and no cough. The heart was beating regularly at the rate of 192 in the minute. It was not enlarged; and the sounds, which were short and sharp, were unattended with murmur. The lungs were healthy. There was no abdominal affection; the urine was free from albumen, and no anasarca was present. Her tongue was a little furred; and her appetite not very good. There was no goitre or exophthalmos.

The patient had been married for three years, and had no family. She said that formerly she was stout; but that for several years

past she had been, as she now was, thin; and that she had been liable for some years past to attacks which seemed to her like that for which she was now under treatment. They came on suddenly, without cause, and after lasting for a few days subsided suddenly. She had consulted medical men, and had been told that she had heart disease, and had been directed not to exert herself, and especially not to hasten up hill or up-stairs. She had never had rheumatism.

On this occasion the heart continued to act with extreme rapidity for a week, and then suddenly its beats fell to 110 in the minute, and shortly afterwards to 92. I saw her again on the 20th of April. The pulse was beating at the rate of 92 in the minute, and quite regularly. There was no cardiac murmur, and she appeared to be, and expressed herself as feeling, perfectly well.

About a month or six weeks later she had another similar attack, in the course of which the pulse reached 246 in the minute. It was unattended with dyspnœa, and there was no rise of temperature.

My second case was admitted into St. Thomas's in July of the same year. It was that of a man between 30 and 40 years of age who had suffered from heart disease ever since an attack of acute rheumatism eight years previously. He had obstructive and regurgitant aortic and regurgitant mitral disease, with hypertrophy and dilatation of the heart. There was reason also to believe that the arch of his aorta was dilated. He presented all the usual signs and symptoms of abundant aortic regurgitation. It appeared that for several months before admission he had been liable to attacks of palpitation; and during the three months he remained under my care he had many such attacks. They came on suddenly at irregular intervals, night or day, without obvious cause, continued for periods which varied between half an hour and thirty-six hours or more, and subsided suddenly. While in his attacks, he complained of feeling ill, faint and weary; and his pulse ranged from 160 to 200 in the minute. In the intervals it varied from 80 to 100. He came under my care for the second time early in 1886, and remained in hospital for a period of four months. For the first two months he suffered, precisely as he had done previously, from recurrent attacks of palpitation, after which I treated him for the first time

systematically with digitalis and iron; and with the best results, for the attacks at once diminished, and during the last six weeks of his residence in hospital, he remained entirely free. He died shortly afterwards; but I have not been able to obtain any information as to the circumstances of his death.

CASE II.—*Double Aortic and Mitral Disease, and probably Aortic Aneurism; Ascites; Paroxysmal Hurry of Heart; Death.*

William N., a dealer, 34 years of age, first came under my care on the 30th of July, 1885.

At the age of twenty-six he had a severe attack of rheumatic fever, and has never felt well since. A few months ago he began to suffer from palpitation, short breath and cough, which often came on in severe paroxysms, and finally prevented him from doing his work.

He was an exceedingly pallid man, complaining of the symptoms above enumerated. The præcordial dulness was extensive. The heart was much enlarged; its apex beating in the 6th interspace, about three inches below, and an inch and a half to the left of the nipple. There was marked pulsation over the whole of this area, and also in the inner part of the right second and third intercostal spaces. The action of the heart was regular, and attended with a musical systolic murmur at the apex, and a well-marked double murmur at the base. There was some crepitation at the lower part of both lungs. The cough was attended with a good deal of frothy expectoration. The urine contained a trace of albumen.

The patient remained in the hospital until the 31st of October, presenting in a greater or less degree the symptoms above described, and suffering also from great distress and weariness, and not unfrequently from pain extending down the left arm. The most interesting phenomenon in his case, however, consisted in the frequent supervention of attacks of palpitation. These came on suddenly, without definite cause, sometimes in the night, sometimes in the day, and ended suddenly, also without definite cause. They lasted from three-quarters of an hour to 36 hours or more at a time; and while present the rate of the pulse (which for the most part continued regular) varied from 160 to 200 in the minute. Most commonly it was about 180. The attacks came on irregularly, sometimes every day, occasionally even twice a day, and often at intervals of several days. During them the patient felt ill, faint and weary, but did not suffer from cardiac pain. He complained of

dyspnœa, but the respirations were for the most part only about 32. He was liable to slight rises of temperature; but there was no obvious relation between them and the palpitation. In the intervals between his attacks the pulse varied from 80 to 100, was generally regular, and presented a well-marked aortic regurgitant character. Various remedies were given with the object of arresting the palpitation. But latterly nitro-glycerine alone was employed, in doses varying from $\frac{1}{100}$ to $\frac{1}{25}$ grain. This was thought to relieve him at times. But I am doubtful whether it had any real influence over the action of the heart.

On the 12th of February, 1886, he was again admitted under my care. He had suffered during the interval from frequent attacks of palpitation, and latterly his belly and legs had become swollen, and he had had severe attacks of epistaxis. His abdomen was tapped on the 4th of March, when 14 pints of serum were removed; on the 27th, when $12\frac{3}{4}$ pints were taken away; and lastly on the 18th of April, when the amount withdrawn was 13 pints. After the tappings the liver was found to be considerably enlarged. The removal of the ascitic fluid relieved the patient's breath, and rendered him generally more comfortable, but had no material influence over the attacks of palpitation.

These continued, in fact, without change of character up to the 13th of April; they were just as severe, just as frequent, and just as irregular in their occurrence and duration as they had been during his former stay in the hospital. On the 13th of April, with the object partly of benefiting the dropsy, partly of influencing the attacks of palpitation, a mixture, containing ten drops of tincture of digitalis, five grains of tartrate of iron and calumba, was ordered to be taken every six hours. This was five days before the last of the three tappings above mentioned. During the next week or two there were two or three short attacks of rapid action of the heart, and then they ceased altogether; so that during the remainder of his stay in the hospital, a period of about six weeks, he continued quite free from them. Moreover, although for a short time the ascitic fluid seemed to be re-accumulating, it soon ceased to collect; and when he left the hospital there was little or none remaining. Nevertheless, for some two or three weeks after the last tapping, although not suffering from palpitation, he was extremely ill, very weak, very drowsy, inclined to ramble at times, and had several severe attacks of epistaxis. Then he improved somewhat, and though weak and ill when he left the hospital, was, on the whole, a good deal better than when he was admitted. During both his residences in the hospital his urine contained more or less albumen, and his temperature varied from

97 to 101. In the intervals between the attacks of palpitation his pulse ranged from 80 to 110, and was apt to be irregular. And after the last tapping his respirations were, on the whole, more rapid than they had been previously, and often rose to 40 or even 50 in the minute.

He left the hospital on the 7th of June, apparently much better than he was when admitted, but still very ill. He died at home three or four weeks later.

My next case was that of a lady, 49 years of age, who for several years had had a largish goitre; but, so far as could be ascertained, had not suffered from palpitation, or other symptoms of Graves's disease. When I saw her she had been complaining for two or three weeks of cough, dyspnœa, and great rapidity of heart's action. It had been ascertained on several occasions that the beats were about 200 in the minute. At the time of my visit she was in bed, complaining of a frequent dry cough and palpitation; but in other respects seemed to be in fair health. The heart (the apex of which was somewhat displaced to the left) was beating at the rate of 180 in the minute, and somewhat irregularly. Its sounds were free from murmur, and no evidence whatever of disease either in the thorax or in the abdomen was discovered. I saw her again three days later. She was then very restless and ill, and her cardiac pulse, which had been 240 the night before, was now about 220. There was still no discoverable visceral disease. The next day her urine was found for the first time to contain a good deal of albumen. She died the following night. I am inclined to think that the goitre in this case was a mere accident of the case, and did not imply the presence of Graves's disease, and to suspect that the attack in which the patient died was only the last of an unrecorded series of such attacks.

Case III.—*Goitre—Rapid Action of Heart—Restlessness—Albuminuria —Death.*

Mrs. P., aged 49, a patient of Dr. Wyman's, of Putney, has, on the whole, had good health; but for several years has had a largish goitre, which is said latterly to have caused some difficulty of swallowing. She has been treated with iodide of potassium,

and, it is believed, with benefit. There has never been an exophthalmos, or stridor, or, so far as is known, attacks of palpitation.

Her present illness began two or three weeks before I was asked to see her, on the 16th of February, 1886; and has been characterised by cough without expectoration, shortness of breath, and great rapidity, with some irregularity, of heart's action. Her illness, however, has not prevented her from performing her accustomed household duties; and it was only yesterday that, by Dr. Wyman's orders, she took to bed. Her pulse has been several times counted, and found to be about 200 in the minute. Her appetite has been poor, but she has slept well, and her urine has been free from albumen.

She was a healthy-looking woman; and, as she lay in bed, seemed free from dyspnœa and all other kinds of distress. The goitre was rather large. There was no lividity or anasarca. Her tongue was clean. She complained of cough, but it was not severe, and there was no expectoration. No evidences of disease were discovered in the lungs. The præcordial dulness was rather large, and the heart apex was thrown a little outwards, but there was no undue prominence. The pulsation was somewhat diffused. The heart was beating slightly irregularly, at the rate of 180 in the minute. The sounds were short and sharp, but free from murmur. No pericardial friction; pulse weak, somewhat irregular. No evidence of abdominal or renal mischief.

I saw her again three days later (the 19th). She was then very ill. It appeared that she had been getting worse, and that since yesterday she had been extremely restless, rarely sleeping, and then only for a few minutes at a time, and constantly rambling. There was no sickness; but she had taken little food, and the bowels had been somewhat confined. Her evacuations had been passed consciously. The pulse last night had been 240. The urine had been free from albumen; the temperature a little above the normal. She was now exceedingly restless, tossing her limbs about, and shifting her position constantly. She was rational, but her articulation was very indistinct. Skin dry, but not hot; tongue thickly coated. Occasional cough, but no marked difficulty or acceleration of breathing. Chest resonant, breath-sounds healthy. Præcordial region as before. No pericardial or cardiac murmurs. Action of heart somewhat irregular, and varying from 200 to 220 in the minute. No paralysis, no affection of pupils, no fits—nothing in fact to point to cerebral mischief. Goitre unchanged.

I heard the next day that the patient was still extremely restless; that her pulse continued about 220; and that she seemed

sinking. The urine now was found to contain a considerable quantity of albumen. She died either that night or the next morning early.

The case of Miss J., which follows, is one of remarkable interest. She was a highly intelligent and active-minded woman, and for three or four years had occupied a post which not only involved continuous official labour and much responsibility, but required her two or three times a year to make a tour of inspection embracing some of the larger provincial towns. Her attacks of palpitation began about the year 1870, when she underwent a long spell of hard work and anxiety, which was followed by a long period of sleeplessness. The first attack came on quite suddenly, and after half an hour ceased suddenly. Subsequently she had many such seizures, coming on at variable intervals, often of many months, and usually without apparent cause. They increased latterly, but rather in duration than in frequency. The attack immediately previous to the one in which I attended her, came on towards the end of 1885, and lasted for six weeks, during the whole of which time her cardiac pulse was regular, and varied from 200 to 260 in the minute; and she continued to do her official work, and generally to act as though she were a healthy woman. Her next attack came on on the 10th or 11th of April, 1886, and ten days later I saw her for the first time. She was then suffering from a troublesome cough, and complaining (as was usual during her attacks) of fidgetiness, and of feeling in a hurry; but otherwise she seemed well, and was performing her official duties as usual. Her cardiac pulsations had ranged between 200 and 250 in the minute, and at the time of my visit were 216. There was no obvious enlargement of the heart; its sounds were free from murmur, and no evidence of disease was discovered either in the other thoracic or in the abdominal organs. At the end of five weeks the pulse suddenly fell to the normal, and for five days continued normal. On the 10th of May, without warning, the palpitation recurred; and, while still suffering from it, she determined to go on one of her periodical tours of inspection. She was feeling very poorly

when she left London on the 1st of June; on the 4th of June, for the first time in her life, her legs became swollen; on the 9th, she reached Liverpool, and feeling very ill, telegraphed for her brother-in-law to fetch her. Nevertheless on that day she spent six hours in inspection. It only remains to say that from this date she suffered from anasarca, pulmonary apoplexy and pleurisy, that the rapid action of her heart continued, and that finally she died with the usual symptoms of cardiac inadequacy on the 10th of July.

It is remarkable that, excepting during her last attack of palpitation, which was unusually prolonged and doubtless aggravated by persistence in the performance of laborious work, Miss J. suffered little while her heart was beating with extraordinary rapidity, and was able to take long walks, and to perform systematic and laborious mental and bodily work. She usually complained of discomfort and worry when the palpitation came on, but soon apparently became reconciled to it, only feeling somewhat irritable and restless. It is remarkable, too, that there was no clear evidence even of cardiac hypertrophy or dilatation.

CASE IV.—*Paroxysmal Hurry of Heart and Restlessness of Fifteen Years' Duration—Death with Symptoms of Cardiac Obstruction.*

I first saw Miss J., aged 40, with Dr. Floyer on the 21st of April, 1886. She had then been suffering for ten days, and was still suffering, from an attack of palpitation.

Her history was as follows:—She had had good health down to the time of the Franco-Prussian war, when, owing to circumstances on which it is needless to enter, she was for some time overworked and had much responsibility thrown upon her. Following on this she suffered for some months from sleeplessness, and had to take chloral. It was about this time that she had her first attack of palpitation. It came on suddenly and left her suddenly, lasting for about half an hour. This attack was followed by others, which seized her at irregular but long intervals, often of many months. These have increased upon her of late, but rather in duration than in frequency. They have come on without warning, and for the most part without obvious cause; but she thinks they have sometimes been induced by dancing and excitment. The last attack occurred in the latter part of last year, and continued

day and night for about six weeks. Like all its predecessors, it began and ended suddenly. The palpitations have always been a cause of distress to her; but have never prevented her from taking exercise or performing whatever duties she has had to perform. For the last three or four years she has been a Government inspector of needlework, and her employment has obliged her to spend some hours daily in official work at Westminster, and periodically to visit various towns in England for the purpose of inspecting the needlework at certain schools; and she has performed these duties thoroughly, even while the attacks of palpitation have been on her, and apparently with injury. What has been the rate of the heart's contractions during the numerous attacks she has experienced I cannot say; but in the last one it certainly varied between 200 and 260 in the minute. The higher number was more than once counted by Dr. Sharkey, who was seeing her at that time. She says that she always feels much distressed when an attack first comes on; but that afterwards she gets accustomed to it, and the distress diminishes; and that she cannot lie on her back or left side on account of the unpleasant throbbing of her heart; but otherwise is unconscious of its rapid action. Her main complaint is that when the palpitation is on her she always feels irritable, fidgety, and in a hurry as if (to use her own expression) she was impelled "to do three days' work in one." She has not been troubled with shortness of breath. She has never had rheumatism, chorea, enlarged thyroid or exophthalmos. She has not been hysterical, and the catamenia have been regular.

Her present attack, as has been stated, came on suddenly ten days ago, and has continued ever since, her pulse ranging the whole time between 200 and 250 in the minute. She has had an irritating cough upon her for some days, which seems to have added to her discomfort; and consequently for a day or two she has remained at home.

She was a healthy-looking woman, apparently not suffering much distress; and indeed no one to look at her or to converse with her would have thought there was anything the matter with her. There was no lividity or puffiness of face, no distress of breathing. Now and then, however, she had a rather violent paroxysm of coughing, attended with little or no expectoration. On examining the chest, the heart was found to be beating at the rate of 216 in the minute, and regularly. Its sounds were short and sharp, and quite free from murmur. The apex of the organ was not displaced; and the præcordial dulness was not enlarged; but the pulsation of the heart was diffused. The lungs were resonant and

the respiratory sounds were normal. There was no sign of abdominal affection. The urine was free from albumen. She was eating and sleeping fairly well. I may mention here that she was a singularly bright, intelligent and well-informed woman; and not only took great pleasure in her official duties, but was generally well-informed, and could talk sensibly and pleasantly, and with piquancy, on many subjects.

The next time I saw my patient was on the 3rd of May. I called on her at her office late in the afternoon. She had been performing her daily duties ever since I last saw her; and now, having been at her post fully employed from an early hour in the morning, was preparing to leave. She looked well, conversed freely and without difficulty, and said that she felt well but for the feeling of unrest, and the impulse to hurry, which were always present when her heart was acting rapidly. The heart was beating at the rate of 208 in the minute; and had been going on thus without intermission for twenty-two days. She still had a little cough.

I did not see her again until the 30th of May. I learnt then that she had awoke on the morning of the 5th of May with her pulse at 120, and feeling quite well: that in the course of the day the pulse had fallen to its normal rate, varying between 70 and 80; and that for five days she continued absolutely healthy. This subsidence of the rapid cardiac action was not due to any obvious cause. She had been working hard and sleeping badly down to the very moment of its occurrence. On the 10th, without manifest reason, the morbid action of the heart recommenced suddenly, and it has continued ever since.

She was not feeling well at the time of my visit; she had been sleeping badly, her appetite had been poor, she had occasionally been retching in the morning, and she had felt weak; moreover she had been suffering from cough. But she had been doing her work thoroughly, and contemplated starting on a visit of inspection to Durham, Liverpool and some other towns. Her heart at this time was beating regularly, and at the rate of 232 in the minute. There was no murmur; the sounds were quite distinct; but there was scarcely any interval between the first and second. The pulse was weak, and could not be counted at the wrist. Her breathing was quick, but I was unable to determine its rate because it always underwent unintentional modification when I tried to count it. But she did not complain of dyspnœa, and spoke as usual freely and without difficulty. There was no lividity or anasarca. The lungs were normal.

On the 1st of June she started alone for the north, as she had

intended, and visited amongst other towns Durham, and lastly Liverpool. She felt ill when she left London, and continued feeling ill during the whole of the time she was away. She suffered from a noisy, distressing and paroxysmal cough. On the 4th of June for the first time in her life her legs became œdematous, and the œdema increased during the rest of her journey. On the 9th she spent six hours in inspecting a school at Liverpool; but she did it with difficulty, and in the evening was brought up to town by her brother-in-law, who had been sent for to her from London.

I paid her a visit on the 10th. She was then extremely ill. Her breath was short; her cough (unattended with expectoration) was very troublesome and violent; she complained of much pain in the præcordial and epigastric regions and across the lower part of the back. Her tongue was clean, but she had loathing of food and frequent sickness; no affection of fauces could be discovered; the lungs were resonant, and the breath-sounds normal; the heart was beating regularly and without murmur, at the rate of 208 in the minute; there was tenderness below the ribs in the hepatic region, and apparently slight enlargement of the liver; and the urine was scanty; her legs were œdematous, and she perspired profusely.

Afterwards she passed a very restless night, suffering much from cough and dyspnœa, and from præcordial and epigastric pain. And when I saw her on the afternoon of the 11th she was much worse than I had yet seen her. She was perfectly sensible; but the præcordial pain, and distress were so great, her breathing was so difficult, each recurring paroxysm of cough was attended with so much aggravation of her sufferings, and she was so collapsed, that her life was believed to be in imminent danger. Her pulse was regular, and 240. There were no abnormal cardiac or respiratory sounds. A few whiffs of chloroform gave great relief to her cough; and in the belief that her sufferings were largely due to over-distension of the right side of the heart and to congestion of the liver, twenty leeches were applied to the præcordial region. These bled freely, and gave her great and immediate relief.

When I visited her on the 12th, I found that she had had a fairly comfortable night, and that she continued much easier. But she was very weak, had occasional attacks of dyspnœa, her cough, though reduced in severity, continued, and her legs were still anasarcous. She did not complain of palpitation, but her heart was beating 240 in the minute. Her respirations were 40. The urine, for the first time, was found to contain a little albumen, but no casts were discovered. The chloroform still relieved her cough.

During the next day or two there was no very great change in her condition on the whole; she had fairly good nights, owing probably to the subcutaneous injection of morphia, her cough was better, but her appetite was very poor, and at times she went off into an alarming state of collapse.

On the afternoon of the 14th she was attacked with agonising pain in the region of the heart—so severe that she was constantly clutching at the left breast with her hand, rocking herself to and fro, and crying out and groaning. She was very pale and ghastly-looking, but not distinctly livid. The pain was continuous, was much like that she had suffered from previously, and did not appear to be true angina pectoris. The inhalation of nitrite of amyl was had recourse to, however, and with some degree of benefit. Chloroform was subsequently employed. And between them, these remedies, and the application of dry mustard to the chest, gave considerable relief. During this time her pulse was 208, and her respirations 40; and for the first time I detected a little crepitation at the base of the left lung. Her temperature was 100·4.

About 7 P.M. Dr. Floyer gave her 30 grains of bromide of potassium, and at 7.40 injected one-eighth grain of morphia, after which she had some sleep. About 9.30 she awoke, and felt what she termed a "lilt" in the region of the heart, or as if "one of its cogs was broken," she became at the same time more faint than she had ever felt before, and thought she was dying. Her sister came to her at once, and found her almost insensible and perfectly quiet, with cold extremities and imperceptible pulse. A minute or two later her brother-in-law arrived. She had already rallied somewhat; but she was still much prostrated, her hands were cold and clammy, and her surface generally perspiring profusely. Her pulse had fallen to 136. She soon recovered from her collapse, and subsequently passed a comfortable night.

On the next day, the 15th, she was much better than she had been for some time. She had little or no distress of any kind; her appetite had improved; her cough had almost subsided; she had no pain; she looked bright; her pulse (which could be easily counted at the wrist) varied between 128 and 132; the temperature was 99; and the urine (which had a specific gravity of 1008) was free from albumen. I thought, also, that the dropsy of the legs had diminished.

From this time down to the evening of the 23rd, the improvement seemed to be maintained, if not to progress. She slept fairly well, and for the first time since her journey to the north was able to lie down; her appetite improved; her pulse varied from 112 to

128; and she "felt at peace." Also the dropsy seemed to be diminishing. But her cough became a little more troublesome; there was some crepitation at both bases, and her temperature was febrile, on one occasion rising to 102. On the 23rd she had seemed as well as she had been during the previous few days. In the evening she took 20 grains of chloral, and slept comfortably for an hour or two. Then she woke up suddenly with a severe stitch in the right side of the chest; shortly afterwards had a very severe and painful attack of coughing; and while it was in progress felt a sudden throb in the region of the heart. The pulse at once jumped to 180 or 185 in the minute; and all the old sense of nausea, distress, and hurry returned. When I saw her on the afternoon of the 24th, she looked ill; her tongue was coated, and her appetite gone; she still had a stitch in the right side, and was breathing 40 in the minute; and her pulse varied between 200 and 208. There was no dulness on percussion over the lungs, but loud pleural friction was audible over the whole front of the right side. Her temperature was 100. Ten leeches were applied to the affected side, and much relief to her symptoms ensued.

For the next fortnight there was no material change in her condition, excepting that during the whole time her cough, which was troublesome, was attended with hæmoptysis. The blood was mixed with mucus, was variable in quantity, but never abundant, and generally brownish or rusty-coloured. It was evidently the hæmoptysis of pulmonary apoplexy, to which the pleurisy also no doubt was due. The pain and friction in her right side subsided, and the sickness and loathing of food diminished. But her pulse continued at the rate of 208 in the minute; her breathing was somewhat rapid; and it was thought there was a little fluid in the left pleural cavity; she complained at times of pains in the hepatic region; the swelling of the legs continued, although it was thought that it was gradually diminishing; and her temperature remained elevated by a degree or two. It may be added, that the action of the heart continued to be regular; that no cardiac murmur was ever detected; that there was no clear evidence that the organ was enlarged; and that the veins in the neck were not dilated.

I was sent for on the morning of July 10th, and reached her at 1.30 P.M. I learnt that she had been attacked during the course of the previous night with extreme dyspnœa, which had continued. When I saw her she was moribund. She was sensible, but disinclined to answer questions; she was breathing quickly with rattles in her throat, moaning and frequently calling out for relief; her face was extremely pale, her extremities were cold and her pulse at

the wrist could not be felt; but her heart was acting regularly, and still about 200 in the minute. She died at 3 P.M.

No autopsy was made.

My fifth case possibly does not belong to the same category as those which precede; at any rate irregularity of cardiac action was as marked a feature as frequency of action, and the palpitation (so far as I know) was persistent. The patient was a spare, healthy-looking, elderly man, accustomed to field sports. He had had, he said, "a sunstroke" a year previously, and had been liable to headaches ever since. Five or six months before I saw him, it was discovered accidentally that there was something wrong with his heart. I saw him on two occasions, and on both found his heart beating irregularly at the rate of between 144 and 148 in the minute. He was not nervous at the time, had no cardiac discomfort, and indeed (excepting for the fear that he had heart disease, as he had been told he had) felt and looked quite healthy. There was no evidence of enlargement of the heart and no murmur. And he assured me that within the last few weeks he had run three miles in twenty minutes without distress.

CASE V.—*Sunstroke (?)—Headache—Rapid Action of Heart.*

Mr. S., a spare, healthy-looking country gentleman, aged 65, accustomed to shooting, riding, and out-of-door exercises, called upon me on the 21st of June, 1886. He has had excellent health down to a year ago, when on a very hot day, being on the sea-shore in command of a Volunteer Artillery Corps, he had what was called a sunstroke. What the symptoms were, beyond severe pain at the top and back of the head, I do not know. He does not appear, however, to have had a fit. This pain continued for three weeks, and since that time he has been liable to frequent recurrences of it. For some time past it has been a dull aching all round the head, but mainly on the left side, where it becomes acute when he sneezes or coughs. It is generally absent at night and in the early morning; but it comes on after luncheon, and increases until bed-time. He sleeps well. He suffers slightly from indigestion, and his appetite is not very good. For two months in the early part of the year he was confined to bed with some inflammation of the right big-toe. This was, so far as I can

make out, certainly not gout. He has suffered from occasional rheumatic (?) pains for years. It is stated that the medical man who attended him when his foot was bad, detected "slight valvular disease of the heart."

At the time of his visit he was feeling very fairly well; and I should have regarded him as being healthy had not I found his heart beating at the rate of 144 in the minute, and somewhat irregularly. He did not appear to be, and said he was not, nervous; and, indeed, his cardiac pulsations underwent no change during the whole of our interview. He was not himself conscious that there was anything unusual in the action of his heart; had no uneasy sensation in the præcordial region, and had no shortness of breath. He assured me, in fact, that within the last few weeks he had run three miles in twenty minutes, without the least distress. There was no evidence of enlargement of the heart, and no trace of murmur. All his other organs appeared to be perfectly healthy. His urine was free from sugar and albumen. I ordered him a mixture containing digitalis, iron, and ammonia, and some *cannabis Indica*, in the form of a pill.

On the 30th I saw him once more. There was no material change. His heart was beating at the rate of 148, and apparently quite regularly; but the beats at the wrist were unequal in force, and occasionally imperceptible, so that the radial pulse felt irregular. No cardiac murmur, no dyspnœa, no anasarca. He complained only of headache at times, lassitude, and impairment of appetite.

Mr. C., my next patient, called on me about a week later than the last. He had consulted me in 1879, at which time his heart was acting with much irregularity and frequency. Although I could discover no definite sign of heart disease, and he complained little of distress, I confess I took an unfavourable view of his case. He got well, however, while taking iron and digitalis. In 1883 I saw him again, when he was suffering from a similar attack; and he informed me that he had had such attacks occasionally ever since I had seen him. When he came to me in June, 1886, he looked, and on the whole felt, well; but his heart was beating with slight irregularity at the rate of 168 in the minute; and he furnished me with a written statement of the number and duration of attacks of palpitation he had had during the previous eighteen months. From that time until his death

in June, 1887, the action of his heart continued irregular and rapid; and although never, so far as I know, attaining 200 beats in the minute, frequently I believe presented a rate of 300 beats per minute for a few seconds at a time. He continued apparently well (excepting for the rapid action of his heart) until about a month before his death, when the ordinary symptoms of obstructive heart disease came on.

Case VI.—*Paroxysmal Hurry and Irregular Action of Heart of Seven Years' Standing—Death.*

Mr. C., a short, healthy-looking, commercial gentleman, about 38 years of age, called on me on the morning of the 28th of June, 1886. He told me that he had been attacked suddenly on the 25th with violent palpitation of the heart, which had continued without intermission ever since. It came on without definite cause. He has felt poorly and disinclined for work, and indeed has taken a holiday from business, but has not felt seriously ill. On presenting himself before me, he looked, and spoke, and acted exactly as a healthy man might do; there was no obvious distress, no hurry of respiration, no congestion or lividity of face, and no anasarca. But his pulse was 168 in the minute, and slightly irregular. The præcordial distress was not more extensive than normal; and the apex of the heart impinged in the usual place. The sounds were short and sharp, and quite free from murmur. There was no further evidence of disease anywhere.

About the year 1875 he had syphilis, followed by secondary symptoms, which have never recurred. In Sept. 1879, he was brought to me for consultation, by Dr. Johnstone English. Two months previously he had begun to complain of uneasiness at the pit of the stomach, supposed to be due to indigestion; this had continued, and it was discovered that his heart was acting with great irregularity. At the time of his visit to me he was pale, he complained of slight shortness of breath, his urine contained a little albumen, and his heart was acting very rapidly and with much irregularity. But I could discover nothing else. He had no cough, no anasarca, the lungs were free from congestion, and the heart was apparently of normal size, and its sounds were unattended with murmur.

I was much puzzled about the case, for there was nothing whatever in the history (excepting the previous attack of syphilis) to suggest a cause for the symptoms, and there was nothing to prove the presence of organic heart disease. It was agreed, however, that

he should abstain from business (which he had been attending to hitherto) for a month, and should take iron and digitalis. He got apparently quite well in a short time.

In November, 1883, he called upon me again. He told me he had had good health, excepting that at irregular intervals he had had attacks of palpitation, which were always relieved by the medicine which Dr. English and myself had prescribed for him. He was now suffering from an attack. He complained of a little shortness of breath in going up-stairs, and that he was conscious of the beating of his heart. But otherwise he felt well, and he looked well. There was no anasarca. The heart was beating very quickly and irregularly, and no murmur was to be detected. No definite cause could be assigned for the attack, unless it was that he had been sitting up late. He was a bachelor, and I suspected his habits were irregular.

When he came to me in the present year, he told me that he had continued liable to attacks of palpitation; and he furnished me with the dates and durations of his attacks since the beginning of 1885, as follows:—March 30th, 36 hours; April 13th, 33 hours; May 19th, 18 hours; July 29th, 57 hours; August 2nd, 4 hours; October 22nd, 46 hours; Nov. 25th, 44 hours; Jan. 6th, 30 hours; Jan. 31st, 22 hours; Feb. 24th, 12 hours. He could not always assign a cause for the attacks, but occasionally he thought they followed a little over-indulgence. They seemed always to be benefited by recourse to the iron and digitalis.

He called upon me again on the 1st of July. He had not been feeling well; but had slept well, and looked well. His pulse was beating between 120 and 130 in the minute, and was very irregular. On the 7th he again paid me a visit. He was much better; and ate, slept, and looked well. But he still complained of a little cardiac discomfort. The pulse was irregular and beat 80 times in the minute; but if it had been acting regularly at its generally prevalent rate, the beats per minute would have been 100. His normal rate of pulse, he told me, was about 60. He had been taking his old prescription. It was decided that he should take a few weeks' holiday on the Continent.

Termination of the Case.—The above case was written out for publication, together with several of the other cases, very nearly 12 months ago. The patient has died since, and I here add a brief statement of his subsequent history.

After I saw him in July, he went to Zermatt for a month. He did not climb, but habitually walked three or four miles a day; and returned benefited in general health by his trip, but still with a rapid irregular pulse.

He called upon me in the latter part of February of the present year, complaining of what he called indigestion. This seemed to consist mainly in an aching pain across the upper part of the belly induced mainly by taking exercise. The heart was beating irregularly, 160 in the minute; and I thought the abdominal pain was due to hepatic congestion. On the 2nd of April I saw him again. He was still complaining of abdominal pain; but in other respects felt, and he looked, well. I examined his heart very carefully on this occasion. Its action was very irregular, presenting two distinct alternating rhythms, each lasting for two or three seconds. In the one the heart was beating at the rate of 80 to 100 in the minute; in the other, at the rate of at least 240. There was diffused cardiac pulsation, but the præcordial dulness was not enlarged, the apex beat in its normal situation; there was no murmur, and the second sound at the left base was not accentuated. On May 4th he paid me another visit. He still had the epigastric pain, and for two days had been suffering from diarrhœa. I again examined his heart carefully. It was beating as before. But on several occasions during the brief periods in which it was beating most rapidly, I counted quite distinctly at least ten beats in two seconds; so that quite certainly, although the heart was not accomplishing more than about 160 beats in the minute, these were frequently at the rate of 300 per minute. After this he went to the Isle of Wight for a few days; but he became very ill there, and returned to London in the middle of May, suffering from great difficulty of breathing, general anasarca, and pain and tenderness in the region of the liver. He was livid, and his urine presented a trace of albumen. Dr. Wilbe was called in to attend him, and I met him in consultation. It is sufficient to say that, although the pulse on the whole became more slow and regular, the usual symptoms of obstructive cardiac disease continued; pleurisy with effusion on the right side, for which he had to be tapped on two occasions, supervened; and he died on the 9th of June. No post-mortem examination was permitted.

My seventh case is in many respects the most interesting of the series. The patient was a young man, whose illness apparently dated from a strain twelve years before. There is some reason to believe that, ever since that time, he had had occasional attacks like that for which he came under my care. This had already lasted three months when I saw him; he had only been laid up, however, for six weeks, and latterly he had had some œdema about the ankles. During

the earlier part of his stay in the hospital, his pulse varied in rapidity, often rising to 250 or 260 in the minute, and often presenting considerable irregularity of action, the beats at such times being apt to present sudden alternations of rate between 240 or 250 and half that number. At this time he complained of sickness, aching across the upper part of the belly, and attacks of faintness, associated with lividity and dyspnœa; moreover, the slight anasarca continued, a little albumen appeared in the urine, and he spat a little blood. After a short time he was treated with iron and digitalis, and much benefit resulted; for although his cardiac pulse continued variable, at times quick and at times irregular, it became comparatively slow after a few days, and then fell to 50 or 60 in the minute. On one occasion it sank to 34. With this change the other symptoms of cardiac disease (anasarca, hæmoptysis, albuminuria, &c.) all disappeared, and he left the hospital apparently well at the end of a month. He thenceforth visited me at intervals as an out-patient. For a month he remained at home, and then resumed his occupation. While thus attending, he reported himself as being on the whole fairly well, but liable to attacks of faintness and palpitation. At the time of his visits to me his heart was often found beating at the rate of only 70 or 80 in the minute; but it was liable to be irregular in its action, and its rate would often rise suddenly to 250 or 260. On one occasion I counted the cardiac beats to be 308 in the minute. On the whole the patient seemed to be going on well, but twelve weeks after he first came under my care, and at a time when he seemed to be in the enjoyment of good health, he died quite suddenly. It was noted during life that the heart was enlarged, and that always when it was acting at a healthy rate there was a systolic murmur at the apex. It was believed, however, that the valves were really healthy. At the autopsy the heart was found to be somewhat large and dilated; but the muscular tissue and the valves were healthy, and the ventricles were empty.

CASE VII.—*Rapid Action of Heart—Sudden Death—Autopsy.*

Arthur W., a draper's assistant, 19 years of age, was admitted into St. Thomas's, under my care, on the 4th of December, 1886.

He has had most of the ordinary diseases of childhood, but never any serious illness. When 8 years of age he took part in a paper-chase, but after running across two or three fields, had to stop suddenly on account of a severe pain in the region of the heart. He leant against a tree, and was speechless for some minutes; but was able to walk home. After that, running was prohibited for a time. He seems to have been liable to palpitation on exertion, especially on running up-stairs, ever since; but has otherwise had excellent health up to three months ago, from which time he dates his present illness.

He was then attacked with general aching pains and sense of poorliness, some cough, and shortness of breath. The symptoms persisted, but nevertheless he continued to follow his employment for six weeks. At the end of this time, his abdomen was swollen, and his legs were œdematous, and his palpitation and dyspnœa had increased. Latterly, too, he has complained of aching across the upper part of the belly, and of vomiting. He has had syphilis.

The patient was a tall, well-made, and, on the whole, healthy-looking man, complaining of sickness, cough with slight expectoration, shortness of breath, palpitation, and swelling of the lower extremities. His face was somewhat pale, with perhaps just a trace of duskiness, and there was slight œdema about the ankles; the thyroid body was not enlarged, and the eyes were not prominent. At the time of admission it was noted that the heart was beating between 240 and 250 to the minute; that pulsation could be felt and seen over the whole præcordial region, and that the cardiac sounds, which were quite distinct, were free from murmur. The præcordial dulness was somewhat extensive, beginning in the third interspace above, and extending slightly to the right of the sternum. The apex impinged in the 5th interspace about half an inch outside the nipple. The lungs were resonant, and, excepting that on deep inspiration some finish crepitation was audible at the right base, the breath-sounds were healthy; expectoration slightly tinged with blood; respirations 32. There was no ascites or evidence of enlargement of abdominal organs. The urine had a sp. g. of 1025, and was free from albumen. Tongue clean, appetite good, bowels regular, temperature normal.

Dec. 5th, 2 P.M.—The patient has had several attacks of

fainting during the morning. They are said to have been attended with dyspnœa, and temporary stoppage of the heart, followed by over-violence of action. When I saw him the pulse at the wrist was irregular and impossible to count. On listening over the præcordial region the heart's action was found also to be irregular and very rapid. For periods of ten seconds or so at a time it was beating at the rate of 200 in the minute; then a strong beat would occur, and the pulse would drop for some seconds to a rate of 108; and again, after a short time, another throb would come, to be followed by renewed over-rapidity. This alternation of very rapid and comparatively slow pulsation continued during the whole time he was under observation. On one occasion the beats fell to 84. At 4 P.M. he was complaining of feeling faint, and his heart was beating at 250 in the minute. His breath was short.

Dec. 6th.—He had several attacks of sense of suffocation and faintness during last evening and night. They came on for the most part as he was dozing off, and prevented him from sleeping. About one o'clock in the morning he was in great distress; very restless, and wanting to get out of bed. He had half a drachm of aromatic spirits of ammonia and ten minims of tincture of digitalis given him, and became easier in the course of half an hour. The heart, when it was examined this morning, was beating at the rate of 164, and regularly. He has been complaining of nausea, and his feet are still œdematous.

Dec. 7th.—Passed a better night; had no return of faintness. When examined this morning the heart's beats were 182 in the minute; at half-past three in the afternoon they were 208. Cough better, expectoration less; urine sp. g. 1025; no albumen.

Dec. 9th.—Improving; but heart beating 224 in the minute.

Dec. 11th.—Seemed better all yesterday; but became poorly during the night; was restless, and suffered from nausea, sickness and diarrhœa. In the early morning the cardiac pulsations were 256; at 11 A.M. the cardiac pulse was declared by himself, and said by the sister of the ward, to be very slow. At noon, however, it had risen to 264. The respirations were then 28, and sighing; and the pulse could not be felt at the wrist; skin dry. *Midnight.*—Was sick at 4 P.M. During the evening has suffered from dyspnœa and palpitation; face now congested, lips and eyelids bluish; skin cool, perspiring; very restless and thirsty; heart's pulsations 256; respirations 28, and sighing; has spat a little bright blood. Ordered fifteen minims of tincture of digitalis.

Dec. 12th.—An hour and a half after taking the digitalis

he lost his lividity; felt easier, and inclined to sleep; but the heart's action remained rapid. Afterwards passed a fair night, and was tolerably comfortable in the morning. At 10 A.M. the cardiac pulse was 200; at 2 P.M. 220; the urine for the first time presented a trace of albumen. Ordered ten minims of tincture of digitalis, five grains of tartrate of iron, with infusion of calumba every four hours.

Dec. 14th.—The patient is much better; no recurrence of dyspnœa or faintness, and no return of hæmoptysis; no albumen in urine. He says that he feels "like his old self." But the pulse is still rapid. In the morning the heart's beats were counted at 168. At 3 P.M. the heart was again examined; it was then acting irregularly; at one time at the rate of 168, at another at that of 192, with short intervals of uncertainty.

Dec. 17th.—The patient has been going on well. The heart, however, has been acting variably, and, on the whole, rapidly. The cardiac beats have ranged from 120 to 132. At the present time the action is generally regular; but occasionally there are series of alternate strong and weak beats, the former only causing perceptible pulsation at the wrist.

Dec. 19th.—Doing well, and feels well. Pulse 60, regular and soft; no anasarca.

From this date until he left the hospital on the 5th of January he continued to feel in his ordinary good health; all faintness and dyspnœa, cough and anasarca, had disappeared; his appetite was good, his urine free from albumen, and he slept well. The heart's pulse was usually regular and about 60 in the minute; occasionally, however, it went down to 52, or rose to 78. On one occasion (December 21st) when I came into the ward the cardiac beats were only 34 in the minute: and the patient, though otherwise feeling well, complained of his heart thumping; consequently, the mixture containing digitalis, which hitherto he had been taking every four hours, was directed to be given thenceforth three times a day. On another occasion, while examining his heart (which was then acting regularly 60 times in the minute), I made him hold his breath for a few seconds, when immediately its action became irregular, presenting alternate strong and weak beats, at the rate (in combination) of 80 in the minute. The irregularity suddenly ceased after some seconds, and the rhythm and rate of movement returned to their previous condition.

When the frequency of the heart's action became permanently reduced, a soft systolic murmur was recognised at the apex. This had never been detected while the heart was acting rapidly, and was never observed to be absent when the organ was beating

slowly. It was perfectly distinct, but was inaudible in the back. All the other cardiac sounds were normal; and the second sound at the left base was not accentuated.

During the times at which the heart was acting rapidly, there was diffused pulsation over the præcordial region. This I examined particularly on two or three occasions, and came to the conclusion that the impulse corresponding to the surface of the right ventricle was synchronous with the apex beat. When the patient left the hospital, the heart's apex was situated just internal to the nipple. The radial pulse, excepting when the heart was beating slowly, was always very small and very feeble, and seemed to be irregular. It did not admit of being counted.

During the patient's stay in the hospital, his temperature rose on one or two occasions to 99, or a little more, and on one or two fell to close upon 95. It usually ranged between 97 and 98·6.

When he left he had gained flesh, and he looked, and (save for the presence of the mitral systolic murmur) might have passed for a healthy man.

After his discharge on the 5th, the patient remained quietly at home for a month or so, and then resumed work as a linen-draper's assistant, travelling every day three or four miles backwards and forwards between his home and place of business; and he came to my ward as an out-patient on several occasions. The following are the subsequent notes that were taken.

Jan. 7th.—Woke up at 6 A.M. yesterday morning with pain in the chest, and palpitation lasting about 10 minutes, and ceasing abruptly with a few forcible beats. Cardiac pulse counted immediately after he had walked up-stairs to the ward, and found to be 264, and regular. A little later it had risen to 288. Shortly afterwards, having rested for a while, the pulse had dropped to 84. Half a minute later it was 116, but irregular. After lying down for a short time it was 72, and regular. No murmur heard while the heart was beating rapidly. Pulsation diffused over whole cardiac area. Ferri Tart. gr. viij; Tinct. Digitalis ♏x; Inf. Calumbæ ℥j; t. d. s.

Jan. 18th.—Has had many attacks of palpitation both night and day, and there is a little return of œdema. At the present moment he feels well, but the heart is beating irregularly. Tinct. Digitalis ♏xv; ex haust.

Jan. 28th.—On the 26th, when getting into an omnibus, had an attack of faintness, lasting a second or two. Thinks he lost consciousness. But he has, on the whole, been much better than he

was previous to the last visit. Indeed, although his heart is beating at the rate of 220 in the minute, he is not conscious of palpitation, and feels and looks well.

Feb. 11th.—The patient has continued much better. But while under examination he complained of palpitation, and of a slight sense of faintness. During his visit the heart was acting regularly on the whole, but with extreme rapidity; on several occasions I counted the beats at 256 in the minute, and on several they varied distinctly between 300 and 308. During the examination he was standing up, and made no complaint. But on looking at him, his face seemed rather dusky, and I made him lie down. After a short time I examined him while he was still recumbent, and found his pulse at the wrist 64; the heart's beats being double that number, and alternately strong and feeble.

Feb. 18th.—Has continued to feel well, palpitation came on as he stood up to be examined, and the heart's beats were then 256 in the minute.

Feb. 23rd.—On this evening I got the patient to attend a meeting of the Neurological Society. He felt and looked perfectly well. His heart was beating regularly, and not rapidly, when he first entered the room. But soon the beats rose temporarily to about 240, and many of the members had the opportunity of verifying the fact. Several also of the members verified the presence of an apex systolic murmur when the heart was acting slowly.

After this he continued apparently very well. On the 26th (Saturday) he was engaged in business till midnight, and went home afterwards. The next morning (Sunday) he got up feeling quite well. He ate a good breakfast, and at one a good dinner; after which he went upstairs and played upon the piano. Suddenly the music stopped, and a fall was heard. On entering the room immediately afterwards, his friends found him lying on the floor dead.

Autopsy on the fourth day after death. Body much decomposed; some old pleuritic adhesions; lungs healthy; pericardium healthy; the heart generally was somewhat enlarged and dilated, and weighed 17¾ oz. Its muscular tissue was soft and flabby (doubtless from decomposition). The valves were perfectly healthy; all the cavities were nearly empty. Examined microscopically the muscular tissue showed no sign of degeneration.

There was no evidence of disease in any of the abdominal viscera.

The nervous centres were healthy, as were also the vagi and sympathetic cords in the neck and thorax.

Besides the above cases I have, since I have been on the look out for them, seen several which are obviously of the same class, but whose significance I should probably have formerly overlooked. The following is one of the most interesting and typical of them :

CASE VIII.—*Paroxysmal Palpitation apparently due to Strain.*

I was asked last February, by a medical friend of mine, to see with him the wife of another medical man whom he was attending. She had had excellent health up to ten days before, and had certainly never suffered from undue shortness of breath, or shown any signs of cardiac disease. At the time referred to, she was lifting a heavy box, when suddenly and quite without warning she experienced a sense of distress in the region of the heart, accompanied with faintness and oppression or difficulty of breathing. Since that time the attacks, lasting individually from a few seconds to a few minutes, have been of frequent occurrence, especially in bed. In bed, indeed, their constant and rapid recurrence has kept her awake all night. It was observed by her husband that during them her pulse was extremely irregular, and it seemed to him that (judging by the pulse at the wrist) the cardiac intermissions were frequently of several seconds duration. I received this history (which was subsequently confirmed) as my friend and myself were driving to the patient's house, and suggested to him that most probably these intervals of apparent inaction of the heart were really periods during which the heart was beating with extreme rapidity, though also with extreme feebleness. When I saw her she struck me as a well-nourished, healthy-looking woman; she had no difficulty of breathing, and was not in any physical distress. Her lungs were healthy and resonant. The præcordial dulness was of normal dimensions, and the heart's apex in the usual situation. Its sounds were healthy, save for the presence of a slight and soft systolic murmur in the aortic area ; there was no evidence whatever of dilatation or enlargement of the heart ; or (excepting the murmur) of any disease either in the valves or in the large vessels. There was no anasarca, and the urine was free from albumen ; the pulse was regular, and about 100 in the minute. But during my interview with her, periods of regularity and comparative slowness of cardiac action alternated frequently with attacks (varying in length between 5 and 20 seconds) of marked irregularity. These generally began with one or two powerful beats, which were followed by beats at the rate of

between 200 and 250 in the minute. No pulse was perceptible at the wrist while the heart was acting thus rapidly. She had no pain at these times, but complained of a feeling of much distress. I saw the lady again at the end of April, and learnt that she had improved since I saw her, that for two or three weeks, while she was at the seaside, she had been almost entirely free from palpitation, and in her ordinary good health, but that during the last week or two (since her return to town) the attacks of palpitation had recurred. She had been taking liquor arsenicalis, but not iron or digitalis. She was at her best when I visited her, felt and looked well, was free from anasarca, had no dyspnœa, and the cardiac murmur, which I had recognised on the previous occasion, had disappeared. She had noticed latterly (as she had done before) that the palpitation came on chiefly at night time, and when lying down; and she lay on the bed while I was with her, in order to bring it on. But only two or three slight attacks of irregularity, lasting for a second or two at a time, occurred. She stated that when lying down she could arrest or prevent the palpitation by placing a cushion or pad under the loins, so arranged as to cause the back to be strongly arched backwards.

A case also, which I evidently formerly misinterpreted, has recently come under my observation in consultation with Dr. Clothier:

Case IX.—*Paroxysmal Hurry of Heart of Fourteen Years' Duration—Death mainly from Bronchitis.*

This patient was an elderly lady living in Wales, whom I had known for 17 or 18 years. She had come up to London on a visit in the latter part of April of the present year. She was then in her usual state of health; but after a fortnight she seems to have caught cold, and to have contracted a sharp attack of bronchitis. I was asked to see her on the tenth day. At that time she was very ill, had much dyspnœa (her respirations were at the rate of 40 in the minute), had a frequent cough without much expectoration, and presented very slight anasarca. The urine was free from albumen. Her chest was resonant, but there was some crepitation at the bases. The cardiac pulsations were irregular, and 224 in the minute, the sounds being free from murmur, and the organ (so far as I could make out) of normal size. The pulse at the wrist was imperceptible. Her temperature was normal, and she was quite sensible. The case went on badly, and she died three days later of a combination of asphyxia and debility.

Between 14 and 15 years ago I was staying at her house in Wales, and was called up at night on one or two occasions, when she appeared to be suffering from what I then supposed to be angina-like attacks, and was, I thought, in great immediate danger. The heart was acting rapidly and irregularly, and I believed that she had serious organic cardiac disease. She recovered from these attacks; and I do not know (although I saw her on the average every year or two) that I ever examined her heart again. I heard, however, that she still suffered, from time to time, from similar attacks to those in which I attended her. But nevertheless she maintained fairly good health, and I was surprised to observe, year after year, that there was no aggravation of her cardiac symptoms. I saw her a little more than three years ago, and she then seemed well and cheerful.

I have no doubt, guided by my last examination of her, that her case from the beginning was a typical one of functional hurry of the heart, and that there never was any organic disease of that organ. Whether she would have died of her bronchitic attack, if her heart had been functionally healthy, may be questioned.

1. The cases just narrated possibly admit of arrangement in several categories. Thus, in some, the action of the heart was only extremely rapid; in some, it was rapid and also irregular; in most, the attacks of palpitation were paroxysmal; and in some the palpitation appeared to be persistent. Undoubtedly the most striking cases (and these first attracted my attention) were those in which phenomenal hurry of the heart, without obvious irregularity, occurred habitually in paroxysms of varying duration, in persons who were apparently in other respects healthy. A wider experience, however, has satisfied me that there is no essential distinction between the cases characterised by simple rapid cardiac action, and those in which rapidity and irregularity are combined, and that, although the tendency to intermission or remission of attacks is a common feature of the disease, the palpitation is always liable, sooner or later, to become indefinitely prolonged.

In those cases in which the palpitation occurs in paroxysms, these come on for the most part without warning, and often without obvious cause, last individually from a few minutes to several hours, or in some instances for several weeks or months, and usually end as suddenly as they began. The

intermissions are equally variable in duration, and, especially early in the disease, may be continued for many months.

In speaking of the rapid cardiac action as being in some cases regular, in others irregular, I have to confess that I use these terms relatively only. In the former class the cardiac pulse during the paroxysms may vary, roughly speaking, from 180 to 300 in the minute, even in the same case; but the changes from one rate to another are sudden, and the persistence of any one rate of beat is relatively considerable. In the other class, the cardiac pulsation is characteristically irregular; that is, small groups of slow and strong beats alternate with small groups of rapid and feeble beats, each group lasting between, say, one and ten seconds. The difference between the two cases is very obvious as a clinical fact, but the irregular pulse is apt at times to become regular, and by simple shortening of the duration of the successive rates of beat, it is clear that what seems a regular pulse would become irregular.

In all cases, as might be supposed, the pulse at the wrist is extremely feeble; and even in cases where the action of the heart seems regular, the regularity usually does not extend to the radial pulse. Indeed, when the heart is acting irregularly, the pulse at the wrist is often imperceptible for many seconds together, so that without examination of the heart it might be assumed that this was motionless during the whole of the time. In fact this disappearance of the radial pulse in the intervals between groups of stronger beats not unfrequently misleads the practitioner in this sense.

2. On what the undue rapidity of the action of the heart in such cases as I have narrated is primarily dependent, is very obscure. In most of my cases no definite cause for the commencement of the disorder could be discovered; and in those cases in which a cause was assigned, it was really doubtful if the apparent cause had anything to do with the matter. In Case VIII. the palpitation seems certainly to have been induced suddenly by over-exertion, and in Case VII. there is good reason to believe that the malady dated from an attack of cardiac distress brought on by running. In my 4th case there is no history of violent muscular exertion, but the palpitation first showed itself at a time when the patient had

much continuous work and responsibility. In another instance, Case V., the symptoms appear to have dated from what was said to be a sunstroke—an attack, the real nature of which is doubtful. In Case VI. there was a definite history of syphilis. On the whole, I am inclined to think that the evidence (such as it is) points to the origin of the malady in either mental or bodily over-exertion.

3. The question naturally suggests itself, as to whether these cases have any relation to hysteria on the one hand, or to exophthalmos on the other. In answer to the first question I may point out that most of my quoted cases, and, I may add, most of the other cases I have met with but have not thought it worth while to record, were in men; and that few, if any, even of the women presented any hysterical history, or manifested characteristic hysterical symptoms. As to the second question, I may observe that although extremely rapid action of the heart is one of the usual phenomena of exophthalmos, I have rarely or never observed such rapid action in that disease as has characterised the cases brought together in this paper, nor have I noticed in it the alternations between excited and normal pulsation which was the striking feature of so many of them. At the same time it is noteworthy that in Case III., in which the cardiac pulsations rose to 240 in the minute, there was actually a goitre. There was no additional evidence, however, of exophthalmos in her case; and I have reason to suspect that the association of the goitre with the palpitation was accidental.

4. One of the most remarkable facts in the history of cases of inordinately rapid action of the heart is the capability which many of the patients manifest of taking active exercise, and of doing their ordinary business even when the palpitation is upon them, and the little distress which the palpitation causes. This was noticeable even in the case of my first patient, who was kept in bed, and at rest, not because she felt ill or incapable of exertion, but because the medical man ordered it. Mr. S. (Case V.) was going about apparently well, and assured me that shortly before I saw him he had (although 65 years of age) run three miles in twenty minutes without suffering. Mr. C. also (Case VI.) not only went about his daily business,

but enjoyed a trip in Switzerland. The most remarkable example, however, of this phenomenon was Miss J., who up to the time at which her cardiac affection induced dropsy and other symptons of cardiac incompetence, lived during her attacks as she was accustomed to live when she was free from them, walked and did important official work daily, and even travelled to various parts of the country in pursuance of her duties as a Government inspector.

No doubt in all cases my patients suffered more or less from symptoms referable to the heart, and in some the symptoms were distressing and serious. They were mainly an uneasy feeling with a sense of fluttering in the præcordial region, shortness of breath, especially on exertion, and faintness; and associated therewith, more or less duskiness or lividity. Angina-like attacks occurred in one instance. One or two patients complained chiefly of a feeling of restlessness. And one (Case VI.), who always gave a very clear and graphic account of her condition, remarked over and over again that when the palpitation was on her she always felt irritable and in a hurry, and as though she must do two or three times as much work in a given time as usual. She, moreover, stated that she always experienced much distress at the onset of her attacks; but that this wore off in great measure after a short time, and that the sense of hurry and of fluttering in the region of the heart alone survived.

5. On the whole the results of my cases have not been satisfactory, and judging from them alone the prognosis of such cases is certainly not hopeful. In one or two the disease is still in an early stage, and what the event will be remains to learn. In two or three the disease has lasted for some years; and the attacks have shown a tendency to increase in frequency and duration. But I am sorry to say that, in large proportion, my cases have already proved fatal.

So far as I know, Cases I. and V. are still in progress; and the 8th case is up to the present time doing well, though not cured. Case III. was fatal, but it is possible that this was not a true example of the condition under consideration; and the 2nd case was also fatal, but the patient was suffering from advanced organic heart disease, with probably an aortic

aneurism, and his death may be fairly attributed to one or other of these lesions. All my other quoted cases have died. Miss J. (Case IV.) died of her disease after it had continued for fifteen or sixteen years. Mr. C. (Case VI.) died quite recently, having been liable to palpitation for about eight years. Mrs. J. (Case IX.) also died very lately, after suffering for fourteen or fifteen years. And A. W. (Case VII.) also died, probably at the end of about eleven years after the first symptoms of disease showed themselves.

Miss J. died with the ordinary symptoms of obstructive heart disease (namely, general anasarca, pulmonary apoplexy, and congestion of the liver and kidneys), which showed themselves for the first time about five weeks before her death, and were apparently induced by overwork at a time when her heart was acting rapidly. Mr. C.'s death was brought on in much the same way. His fatal symptoms, which included general anasarca, hydro-thorax (for which he was tapped twice), congestion of liver and kidneys, lividity and orthopnœa, appeared only about three weeks before he died. Williams died suddenly at a time when, so far as his feelings and appearance were concerned, he seemed to be perfectly healthy. Mrs. J. succumbed to what appeared to be a not very severe attack of bronchitis, the effects of which were obviously aggravated by the weakness of her heart.

Speaking generally of these cases of recurrent palpitation, I should be inclined to say, that the prognosis is fairly hopeful for those persons who are able to lead quiet lives, who avoid mental or bodily excitement and overwork, who protect themselves from catarrhal and other disorders likely to interfere directly or indirectly with the equilibrium of their circulatory organs, and who nurse themselves with care during their attacks of palpitation. And I am disposed to think that, in many such cases, the progress of the affection may be arrested. I base this opinion partly on the apparently complete restoration of such patients to health in the intervals between their attacks, partly on the long duration of some of their cases without the development of any serious cardiac and other complication, and partly on the circumstances of their fatal attacks when these at length happen to supervene. But it is

obvious that patients thus affected run many and continuous risks, and that death, either from a sudden faint or from the coming on of symptoms of cardiac incompetence or obstruction, is always imminent.

6. What the condition of the heart is in these cases is a matter of considerable interest. That such attacks of palpitation as I have described may be associated with organic heart disease is obvious, from the history of my second case, in which the patient was suffering from advanced aortic regurgitation combined with hypertrophy and dilatation of the left ventricle; and from a case now under my care in which the palpitation, coming on for the first time three or four months ago, is associated with the presence of a præsystolic apex murmur, referable apparently to the effects of an attack of acute rheumatism which occurred more than fifteen years ago. But in most of the cases I have seen there has been nothing to show that the heart was structurally affected; there has been no murmur, there has been no accentuation of the second sound at the left base, and the apex has impinged at the usual spot, or so near as it shows that there can have been little if any real change in bulk. In the only fatal case in which I have obtained a post-mortem examination, the heart was somewhat hypertrophied and dilated, but the valves were absolutely healthy, and so also was the muscular tissue. In this case a systolic apex murmur was recognised during life, when the heart was beating slowly, and appears to have been due (as was suspected during life) to the mitral valve allowing of regurgitation in consequence of the ventricular dilatation.

My belief is that the affection has no special connection with cardiac disease, and that dilatation and hypertrophy (when they occur in it independently of valvular mischief) are the slowly developed consequence, and not the cause, of the functional disturbance.

In connection with this subject of the condition of the heart, it is noticeable that during the attacks of palpitation the cardiac pulsations are visible over the whole precordial region, and that there is visible throbbing in the large arteries of the neck. It might be hastily assumed that this extensive and apparently violent pulsation implied undue force of cardiac

action. But that this is not so is obvious, both from the extraordinary feebleness of the pulse at the wrist, and from the very small amount of blood which is pumped at each ventricular systole into the arteries. The explanation is probably that the cavities of the heart are full of blood, and that the contractions of the ventricles are imperfect, so that only a small proportion of their actual contents is expelled at each systole. For if partial contraction occurs in a full but flabby and elastic-walled cavity, as the heart under such circumstances may be assumed to be, it is clear that the contraction in one part or zone must be attended with a general rise of pressure in, and consequent dilatation of, every other part, in which also there would be general synchronous pulsation. I believe, from having carefully examined several of these cases, that the apex beat and its impulse over the cardiac area are actually synchronous; whereas if, as one sometimes witnesses, and especially in cases of aneurism of the ascending arch, the pulsations of the surface of the ventricles and of the apex in a healthily acting heart are visible, they are distinctly alternate in rhythm.

7. As to the real nature of the disease which my paper is intended to illustrate, I have little to say. My belief is, as will doubtless have been gathered from all that precedes, that so far as the heart is concerned it is a purely functional disorder, that any actual cardiac disease which may be present in any case must be regarded as accidental, and that the slight hypertrophy and dilatation of the heart which may be found in patients who have suffered from the malady for years are (as I have already remarked) the consequence, and not the cause, of the palpitation.

8. I have also little to say under the head of treatment. In one or two cases during the attacks of palpitation, I have found digitalis, or digitalis combined with iron, of exceeding value; but in others these drugs have had no beneficial effect whatever. Digitalis and strophanthus, however, are remedies which would naturally suggest themselves, and are always worth a trial. If symptoms of obstructive cardiac disease become developed, as I have shown they are likely to do sooner or later, of course the ordinary treatment for such symptoms should be carried out. The little apparent suffering which

many patients experience during their attacks permits and encourages them at such times to go about their ordinary work and even to undergo considerable mental and bodily exertion. It is clear, however, that all such labours are detrimental, and attended with danger; and that the patients should be kept at rest. When one considers the tendency there is in these cases for their malady to become more and more grave and intractable as time goes on, the importance of treating them in the intervals between the attacks, and especially in the earlier stages, becomes obvious. I am not prepared to recommend at such times the employment of any particular drug, but it seems to me that our aim should be to maintain the patient's general health, to prevent as far as possible undue mental or emotional exertions, or severe muscular strain, or continuous mental or bodily hard work, and to treat with special care every attack of palpitation.

ON RETROBULBAR INCISION OF THE OPTIC NERVE IN CASES OF SWOLLEN DISC.

BY ROBERT BRUDENELL CARTER.

I AM desirous to bring before the profession an account of an operation I have recently performed, by opening the sheath of the optic nerve behind the eyeball, in order to permit the escape of fluid in a case of great swelling of the termination of the nerve within the eye.

An operation for this purpose was first suggested by Dr. de Wecker, who, at the London meeting of the Quadrennial Ophthalmological Congress, in 1872, read a paper on the subject. He pointed out that the investigations of Schwalbe, continued by H. Schmidt, Manz, and others, had shown that the liquid contained in the arachnoidal space may in cases of exaggeration of intracranial pressure be forced through the optic foramen between the two coats of the optic nerve up to its insertion into the eye. Here the liquid, meeting an obstacle in the sclerotic ring, produces distension of the external coat on the one hand, and on the other, strangulation of the contents of the nervous sheath (fibres and vessels), which would explain the disturbance of vision, and the secondary atrophy of the nerve itself. Dr. de Wecker did not enter into any discussion as to the applicability of Schwalbe's theory in all cases, or as to whether the dropsy of the optic nerve is constant in every case of confirmed neuro-retinitis. He maintained it to be indisputable that in the great majority of necropsies the distension of the external sheath near the eye has been met with, and that Schwalbe's theory affords the most satisfactory explanation of the production and symptoms of neuro-retinitis. According to this theory, he considered that there were two indications to be fulfilled: in the first place, to give issue to the accumulation of the cerebral liquid by making an incision into the external coat of the optic nerve; and, secondly, to relieve the strangulation of the nerve by incising the sclerotic ring where it forms the junction of the sheath with the external enveloping membrane of the eye. By proceeding thus, he hoped to relieve the symptoms of compression, not only of the nerve itself, but those of the

cerebral centres—in other words, to remove the pain and inconvenience arising from the excess of intracranial pressure.

Dr. de Wecker went on to say, that he had performed the operation in two cases of cerebral affection (probably tumours of the cerebrum). In one of the cases, a man of 40 (whose right eye was operated on), neuro-retinitis was completely regressive in the right eye, and in the other regressive also. There was only a slight degree of vision, scarcely allowing the patient to do any work, even if the weakness of the legs, with which he was at the same time affected, had permitted it. The other patient, a woman of 30 (whose left eye was operated on), was completely blind, and was at the same time the subject of paraplegia, partial paralysis of the left facial nerve, and great difficulty of moving the tongue. She also suffered from incessant headache. The neuro-retinitis was in this case evidently regressive in both eyes, and especially in the left.

Dr. de Wecker described his operation by saying that he made an incision between the rectus externus and the rectus inferior muscles, at a distance of one centimetre from the margin of the cornea. Then, cutting the conjunctival and sub-conjunctival tissues, he penetrated between the eye and Tenon's capsule with a pair of probe-pointed scissors until he reached the optic nerve. He then introduced a spatula, and luxated the eye upwards and inwards. It was easy after the luxation of the eye to feel with the spatula the distended nerve, and to introduce a sheathed neurotome, an instrument made by Mr. Mathieu for the purpose. It consists of a stem curved to fit around the eyeball, terminating in a notched extremity intended to feel for and to embrace the nerve, and concealing a sheathed blade. Dr. de Wecker believed the mechanism of this instrument to be such, that by slight pressure on the spring only that part of the nerve would be cut with which the notch was placed in contact, at a distance of one centimetre behind the eye. The operator was to incise the sheath of the optic nerve and the sclerotic ring, moving and pressing the instrument from behind forwards. He would then remove the instrument, after having pushed the spring back, so that the blade could not cut more than was desired.

Dr. de Wecker stated the results of his two operations as follows:

1. There was no pain after the operation, which was performed without the aid of anæsthetics. 2. Very great relief of the headache, especially on the side operated upon. 3. On removing the bandage, twenty-four hours after the operation, there was a very slight congestion of the eyeball, and the pupil deviated to the opposite side of the penetrating wound, probably in consequence of rupture of the ciliary nerves.

This deviation of the pupil disappeared in a few days. 4. The operation having been performed in complete regressive forms of neuro-retinitis, no amelioration of sight was to be expected; nevertheless the male patient seemed to have improved in the operated eye, for in a week he was able to perceive the light. The beneficial result in this case seems to have been indisputable, and the medical men who assisted at the operation were of the same opinion, as there was a marked influence on the headache, and on the general state of the health. The male patient was able to stand more firmly on his legs, and to answer questions promptly. He seemed delighted with the operation, on account of the general relief he had experienced, although he had gained nothing as far as sight was concerned. The result of the operation was less manifest in the female patient, who was only relieved of her headache.

The ophthalmoscope did not discover the incision, which was perhaps not made deeply enough; but there was a slight increase in the volume of the vessels, especially of the veins.

In September 1872, Dr. de Wecker's experiment, if I may so describe it, was repeated by Mr. Power on the right eye of a girl of 13, this eye having been blind for two years, and having still some swelling of the optic disc. Mr. Power has favoured me with the notes of the case; but it is sufficient to say that, beyond a doubtful increase in the power of perceiving light, no beneficial effect was obtained.

Dr. de Wecker's paper greatly impressed me when it was read; but I could not escape from the conclusion, that a case of advanced optic-nerve atrophy, although possibly suitable as a *corpus vile* on which to demonstrate the practicability and harmlessness of the operation, was not one in which any good result could reasonably be expected. I felt, moreover, that the mode of procedure was extremely faulty, and that the structures concerned were far too important to be incised without the aid of sight. The least obliquity of direction of the unsheathed blade might cause it to divide one or more of the ciliary nerves; and, even if the blade were placed with diagrammatic correctness, the smallest excess of penetration might wound the central retinal vessels in the nerve trunk. The attempt to extend the incision through the sclerotic ring seemed to be especially perilous, and likely to lead to puncture of the choroid and to extravasation of blood within the eye in the neighbourhood of the macula lutea. In short, the operation, as described by Dr. de Wecker, appeared to me to be too uncertain and too dangerous to be justifiable in any circumstances, although I had no doubt of the soundness of the principle on which it was based.

During the last few years it has been my lot to see a large

number of cases in which, in connection with the growth of intra-cranial tumours, or with other morbid processes, there has been great swelling of the intra-ocular extremities of the optic nerves. In many cases of this kind sight is at first absolutely unaffected, the swelling being limited to the connective tissue of the disc and of the fibre layer of the retina, and being apparently produced by a moderate amount of mechanical impediment to the circulation, not enough either to arrest conduction through the nerve fibres, or to close the channels of the vessels. In some instances the swelling of the optic discs has disappeared under treatment, and complete recovery has ensued. In others, nerve atrophy and blindness have been produced; and these consequences may, I think, be explained in two ways. If I may dwell for a moment upon the anatomy of the parts concerned, it will be to remind the reader that the optic nerve is closely invested by a sheath derived from the pia mater, and this again, but loosely, by a sheath derived from the dura mater. Between these two sheaths there is an interval, or inter-vaginal space, which terminates in a cul-de-sac at the level of the back of the eyeball, where the dural and pial sheaths unite to blend with the sclerotic. This inter-vaginal space contains membranous processes derived from the arachnoid, and communicates with the sub-arachnoid space; so that, as was shown by the authorities cited by Dr. de Wecker, an excess of sub-arachnoid fluid will make its way into the intervaginal space, compressing the nerve trunk and distending the dural sheath, which, especially in the immediate neighbourhood of the eye, has often been found to be much dilated. The central artery and vein of the retina pierce the sheaths at a point which is from fifteen to twenty millimetres behind the eye: and it is manifest that compression of the lower part of the nerve trunk, in which the vein is still contained, must impede the return of blood, and must occasion dropsical swelling of the nerve termination within the eye, and of the adjacent retina, with turgescence of the retinal branches of the veins. The effect of such compression must depend upon its degree, and experience shows that it may be sufficient to produce distinct dropsical swelling, and that this may continue (within my own knowledge for more than three years) without any affection of the sight. On the other hand, the compression of the nerve trunk as a whole may increase so rapidly and to such a degree as quickly to destroy vision by arresting conduction through the fibres; in which case it must also arrest the flow of blood through the vein, an occurrence usually attended by hæmorrhages into the retina. When the compression increases more slowly, the consequent dropsical effusion may excite inflammation of the connective tissue of

the optic disc by mechanical disturbance of its structure; and this inflammation may in its turn give rise to an interstitial plastic exudation, which will gradually contract, and will produce atrophy of the disc by strangulation of its capillary vessels and of its nerve fibres. With the commencement of contraction vision begins to suffer, and is ultimately completely extinguished.

It is very common to hear all cases of swelling of the intra-ocular termination of the optic nerve described as "optic neuritis," but I am strongly of opinion that this description is erroneous. I am only too familiar with optic neuritis; and the first symptom of its existence is great impairment of sight, which, if the inflammation be not relieved, speedily passes into blindness. The neuritis is usually retro-bulbar in its origin; and some days may elapse, and central vision may be almost lost, before any evidences of exudation can be discovered in the disc. Even then, such evidences are often so slight that only a practised eye would detect them; and I presume that the part of the nerve primarily affected is in such cases posterior to the point at which the retinal vein emerges from the sheath, and that hence no mechanical or dropsical swelling of the disc is produced. I do not believe in the possibility of any true neuritis without marked derangement of the function of the affected nerve; and when I see, as I have often seen, considerable swelling of the optic nerve within the eye, continuing for weeks, months, or even years, without any injury to the sight, I feel convinced that such swelling is not neuritic, but only passive or dropsical, a result of impeded circulation. I have no doubt that dropsical swelling may excite a sort of spurious neuritis, in the way already mentioned, by the disturbance which the swelling occasions, but I regard this effect as being confined to the connective tissue, and not as constituting neuritis in any true sense; nor do I think that the mere presence of leucocytes in the optic disc affords evidence of any action to which the word "neuritis" can properly be applied. We have all seen cellulitis of the leg; and we have all seen a certain amount of inflammation, attended by erythema, as a consequence of dropsical swelling; but we should not describe a limb which presented the latter conditions as an "inflamed leg," even though it would no doubt contain leucocytes in abundance, and though the fluid in the meshes of the connective tissue were coagulable and plastic in its character. Notwithstanding all this, we should know that a few judiciously placed punctures, by allowing the fluid to escape, would speedily produce a change for the better; while, in actual cellulitis, not even free incisions would generally prevent destruction of tissue. To confound the two

conditions, on account of the presence of microscopic products common to both, would be to permit histological refinements to outweigh the evidence of broad clinical facts.

The cases to which I am now chiefly desirous to attract attention are those in which, after the persistence of optic nerve swelling for a certain time, without impairment of vision, such impairment at last commences, and proceeds more or less quickly to complete blindness. In some of these cases the loss of sight may be due to the development of a tumour or other intercepting lesion between the optic nerves and the cerebral centres of vision; but more frequently, in my judgment, it is due to one of the two causes I have mentioned, that is, either to rapid compression of the nerve trunk as a whole by increase of fluid within the dural sheath, or to slow strangulation of the capillaries and fibres by the contraction of an interstitial effusion of a plastic character. In some of these cases the primary lesion may be recovered from, in others it may not destroy life for a long period; but in both the patient is left blind.

I may mention, by way of illustration, the history of a gentleman who first consulted me on the 30th of September, 1885. He had then great swelling of both optic discs, but scarcely any affection of the sight. He continued much in the same state until the following April, when his sight began to fail, the previously existing swelling passed into atrophy, and in a few weeks he became totally blind. There is reason to believe that he has an intracranial tumour; but he is still in possession of his mental faculties, and although reduced in strength, has no paralysis. In thinking over his case, I determined that I would take the next opportunity of a similar kind to open the nerve sheath behind the eye, in the hope that the evacuation of the contained fluid would preserve the nerve from atrophy and the vision from destruction. Such an opportunity was afforded by the case which forms the subject of this paper, and which I will now proceed to relate.

E. O., a lady's-maid, 26 years of age, was brought to me by her mistress on the 18th of November, 1886. She had been somewhat out of health for a fortnight or so, had recently been exposed to cold when driving, and had received a blow, of no great severity, on the right parietal region of the head; but there was nothing very definite in either her history or symptoms. She had suffered for ten days from a good deal of aching pain across the forehead, and also in the occipital region, but her chief complaint was that, when rising on the morning of the 9th, she discovered that she saw imperfectly with the left eye. She could distinguish objects, but said that they looked dark and dim, and this state of the sight, although

varying a little from time to time, had remained substantially unaltered. Central vision of the left eye was found to be equal to about two-thirds of the normal standard, but the eye was blind as to the temporal half of its field of vision, and the field was much contracted in other directions also, as shown by the chart in Fig. 1, which was taken on the 26th of November. The left optic disc was swollen, and its margins were entirely concealed, the swelling encroaching upon the retina in an unusually broad zone of circumneural turbidity, through which not even the colour of the choroid could be seen. The central part of the swelling showed numerous small hæmorrhages, and its apex was best defined with a convex lens of four

Fig. 1.

dioptres, which, allowing for one dioptre of hypermetropia, pointed to an actual elevation of one millimetre. Beyond the swelling, the retinal veins were distended and tortuous. The right eye was normal in aspect and vision, and had one dioptre of hypermetropia. There were no general symptoms of a kind to throw any light upon the essential nature of the lesion. The tendon reflexes were active, and there was a trace of ankle clonus.

The patient was admitted into the National Hospital in Queen Square, under the care of Dr. Hughlings-Jackson and myself, and she was placed upon iodide of sodium with mercurial inunction. She very soon became mercurialised;

but the only other obvious change in her condition was that the swelling of the optic disc, and the breadth of the turbid circumneural zone, continued steadily to increase. Some opaque white patches appeared among the effusion, a considerable hæmorrhage occurred over the position of the inner margin of the disc, and on the 17th of December, flocculent opacities in the vitreous were observed over the apex of the swelling.

The vision was at this time unchanged, but I felt that it was exposed to great risk from increasing pressure or from interstitial strangulation, altogether independently of any unknown lesion by which the origin of the local condition might be explained; and I determined, with the assent of Dr. Hughlings-Jackson, to repeat Dr. de Wecker's operation, if possible, in a more certain and satisfactory way. I convinced myself, by trial upon the dead subject, that the insertion of the optic nerve could only be exposed from the outer side of the orbit, and, after much consideration, I determined on the course about to be described.

On the 28th of December, the apex of the swelling being then best defined by a convex lens of nine dioptres, shewing an actual elevation of close upon three millimetres, or twice the diameter of the disc itself, the patient was placed under chloroform, and the lids were widely separated by a rack speculum having its bar on the nasal side. I divided the conjunctiva and sub-conjunctival tissue in a vertical line, about a centimetre from the corneal margin, so as to expose the external rectus muscle a short distance from its insertion. I passed two strabismus-hooks under the muscle, and then, being provided with two threads of fine carbolised catgut, each carrying a needle at either end, I passed the two needles of one thread through the tendon, from within outwards, between its insertion into the sclerotic and one of the hooks, and the two needles of the other thread, also from within outwards, between the belly of the muscle and the second hook, and then divided the muscle between the hooks. The eyeball was next rotated inwards, while the orbital portion of the divided external rectus was lifted and turned outwards. By a succession of small scissor snips, I carefully divided the capsule of Tenon, and other resisting structures, to the necessary extent, and presently succeeded in bringing the insertion of the optic nerve into view. I had furnished myself with a fine sharp-pointed knife, and with a very delicate sharp hook, each mounted on a flexible platinum stem. I picked up and steadied the nerve sheath with the hook, and incised it with the knife, in the direction of the axis of the trunk, for perhaps a quarter of an inch, up to the insertion into the eye, but

without dividing the sclerotic ring. An escape of fluid showed the division of the sheath to be complete. As soon as this was seen, the eyeball was replaced, the parts of the divided external rectus were brought together by the sutures already in position, and the conjunctival wound was united by two others. Both eyes were closed and bandaged, and the patient was put to bed.

The operation was followed by no discomfort. There was no rise of temperature, the muscle united readily, and the external wound showed no irritation. On the next day the patient was somewhat nervous and excited, but on the third day she said that she felt quite well, and that she was completely relieved of the pain and "muzzy feelings" in the head by which she had for some weeks been troubled.

A mere glimpse with the ophthalmoscope, four days after the operation, showed that the swelling of the optic disc had diminished, but no complete examination was made until the 7th of January. By that time the broad zone of circumneural retinal opacity had disappeared, and the margin of the disc was visible around rather more than the outer half of its circumference. The best definition of the apex of the swelling was obtained with a convex lens of four dioptres, showing that the actual prominence had diminished by about one-half, or to what it was when first examined.

From this time forward the subsidence of the swelling, although not rapid, was steadily progressive. The remaining blurring of the inner margin of the disc has disappeared; and all the extravasated blood has been absorbed. The vessels of the disc are visible throughout the whole of their course, and there is no longer any impediment to the venous circulation. There is about one-third of a millimetre of remaining swelling, and the general aspect of the surface is still somewhat veiled and abnormal, but it seems to be in course of steady restoration to the natural condition. If I had divided the sclerotic ring, the recovery might possibly have been more rapid.

The acuteness of central vision has become normal, and early in March the field began to increase on the previously blind temporal side. The chart in Fig. 2 was taken on the 21st of March, in a very bad light, and the field has since undergone further extension.

The movements of the eyeball, and those of the pupil, are perfect, and there is nothing to show that any surgical interference has taken place. The general condition has lately undergone steady improvement, and there is, I think, reason to hope that complete recovery will occur.

The issue of this case seems to me to establish that the

operation devised by Dr. de Wecker, and which, as he endeavoured to perform it, I should not care to attempt, has been brought within the limits of safe and prudent surgery. The case shows that the sheath of the optic nerve can be exposed to view, that any fluid which it contains can be evacuated by incision, and that this can be accomplished without risk to the patient, or without the possibility, assuming the exercise of due care and skill, of any injury to the eye. If thus much be conceded, we have only to inquire in what circumstances, if in any, such a proceeding is likely to be advantageous to a patient.

For the purposes of such an inquiry, I should in the first

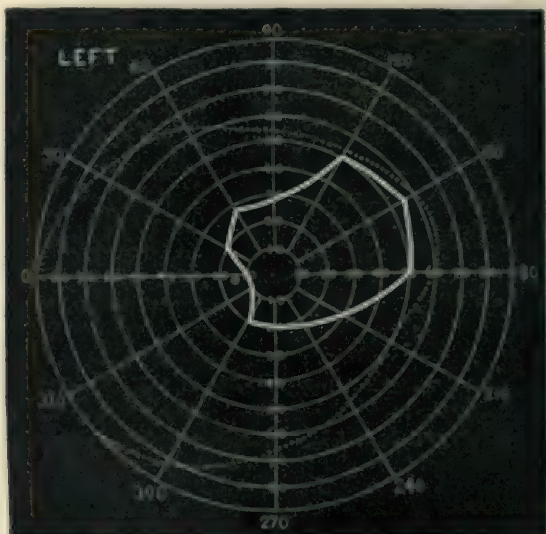


FIG. 2.

place exclude all cases in which swelling of one or both optic discs is apparently due to the presence of an intracranial tumour which can be localised and removed. Mr. Horsley's operations have shown that, in such circumstances, the removal of the tumour itself is followed by the subsidence of the disc swelling; and the results of the major operation would render the minor one unnecessary. But, apart from these cases, I think incision of the nerve sheath should be practised whenever swelling of one or both discs is attended by the commencement of impairment of vision. Such impairment might be caused, in some instances, by a lesion unconnected with the sheath, which its incision could not touch, but it would more

frequently be caused by external compression or internal strangulation of the nerve, and these conditions might certainly be relieved. It is well known that many cases of swollen disc go on to atrophy and blindness while the patient recovers, or at least does not die; and I feel strongly that, in most of these, sight might be preserved by the means to which it has been my object to direct attention.

We also see instances of loss or great impairment of sight, from nerve atrophy after various acute diseases, fevers, the exanthemata, and others, and in these we generally find ophthalmoscopic evidence that the atrophy has been preceded by swelling, probably as a consequence of some meningeal effusion. If it were the custom to watch the optic discs in all grave cases of this kind, especially on the occurrence of head symptoms, the commencement and the increase of swelling would be observed, and incision of the nerve sheath would not improbably preserve the sight in many instances.

I should by no means be without hope, moreover, of obtaining good results from the operation in a certain proportion of cases of real or primary optic neuritis, those of which I have already spoken as being speedily destructive to sight, and as being attended with a very small amount of disc swelling. In these, I apprehend, the neuritis is not only retro-bulbar, but also posterior to the emergence of the retinal vein; and it is highly probable that the inflamed and swollen nerve is compressed and injured by its unyielding sheath. Assuming genuine neuritis to exist, and the nerve to be suffering compression in the manner supposed, I cannot doubt that the relief of the compression might lead to the resolution of the neuritis; and I should be quite prepared, in any such case, to undertake the operati n. I should introduce, through the first aperture in the sheath, a fine probe-pointed knife between the sheath and the nerve trunk, and should run this knife as far back as possible towards the apex of the orbit, in the hope of making an incision sufficiently long to relieve compression over the whole of the inflamed portion of the nerve. In the indicated conditions, and possibly also in others, I cannot but think that the operation I have described has a fair prospect of usefulness; and I beg leave to commend it to the attention of all those who are likely to have opportunities of testing it in practice.

Clinical Cases.

CASE OF MYXO-FIBROMA OF THE FIFTH DORSAL NERVE EXTENDING ON TO THE SPINAL CORD.

BY J. MITCHELL BRUCE, M.D., F.R.C.P.,

AND

FREDERICK W. MOTT, M.D., M.R.C.P.

GEORGE L., aged 37, by occupation a game-keeper, was first seen by Dr. Bruce on May 7th, 1886, for incomplete paraplegia with impairment of sensation as high as the epigastrium, some difficulty of urination, and constipation:

He complained of a boring pain in the mid-dorsal region, radiating into the left axilla, and of a feeling of numbness in the left lower limb, extending upwards. The pupils were equal, and reacted both to light and accommodation. The knee-jerk was well-marked on both sides. Physical examination of the chest revealed nothing abnormal in the condition of the thoracic viscera, nor was there any evidence of disease of the abdominal organs. The patient was recommended to go to Charing Cross Hospital, and on May 9th he was admitted under the care of Dr. Bruce. The following notes were taken:—

With the exception of a brother having died of phthisis, the *family history* was good. He was married when 27 years of age, and has had four children; one only is living. There was no history of syphilis. Until four years ago, patient had been in robust health. Many years ago, he had had a fall on his back; but he did not think that there was any permanent injury resulting from it, for many years elapsed between this and the period when symptoms of his complaint appeared.

It was about four years ago that he noticed a sharp pricking pain in the fifth intercostal space of the left side, just inside the nipple. This continued, and, after a month or so, became more severe and stabbing in character. For about three years this pain persisted; but he was able to continue his

work until Christmas 1885, when, on account of the great pain he experienced in walking, he was obliged to give up his occupation as a game-keeper. The pain was particularly severe if he bent down; for example, to tie his boot-laces.

Just after Christmas he noticed that his legs swelled, and the following March the left foot began to feel numb, the numbness gradually spreading up the left leg. He noticed this also, though to a much less extent, in the right foot. On account of this numbness in his feet, patient says he "blundered about." In less than a week after the numbness had set in, he noticed that his legs, which were usually extended during sleep, often became flexed. He said that he had felt weakness for some time in his left leg, and since Christmas he had been troubled with constipation and difficulty in micturition. He has had no priapism nor loss of sexual power. The reason he sought admission to the hospital was for loss of power and numbness in his legs. He believed that the pain he had suffered for four years had little to do with his present illness, having been told by the doctor it was rheumatism.

May 12th.—The patient now complains of numbness in the *right* lower limb, increasing in degree. Plantar reflex distinct; cremasteric and abdominal reflexes absent; knee-jerk increased, especially on the left side. Ankle-clonus on the left side.

May 15th.—Patient complains of pain in the mid-dorsal region which radiates round the thorax, especially on the left side. Occasionally the pains shoot down the left thigh; these pains are particularly severe at night. There is a girdle sensation at the level of the fifth intercostal space. Absolute anæsthesia and analgesia exist over the lower limbs, and on the trunk up to the level of the umbilicus. Power of movement of lower limbs is now completely lost on both sides. Occasionally there is spasmodic flexion.

Reflexes.—Plantar reflex still present; cremasteric and abdominal, lost; epigastric, present; knee-jerk, lost; ankle-clonus, now absent.

There is complete loss of control over the rectum, retention of urine, and unconsciousness of rectal and vesical acts. Œdema of the lower limbs, and bedsores on the hips.

May 28th.—Condition carefully investigated. Anæsthesia as high as the eighth rib; above this to the fifth intercostal space is a zone of hyperæsthesia. On the right side, there is anæsthesia as high as Poupart's ligament. Painful sensations are lost as high as the margin of the ribs on the left side, as high as the middle of the thigh on the right side. Sensations of heat and cold are not appreciated on either side

below the level of the umbilicus. Plantar reflexes, exaggerated; slight ankle clonus on left side; knee-jerks, absent; cremasteric, epigastric, and abdominal reflexes all absent.

Muscles.—Right limb: can flex and extend ankle, knee, and hip joints, but there is considerable weakness. Left leg completely paralysed at all the joints except the toes, where there are slight voluntary movements. Occasionally the legs extend themselves; and sometimes patient finds them flexed on waking. No rigidity exists at any of the joints. There is slight redness and swelling of the left knee-joint, with some effusion. The muscles of the limbs are not wasted; and although requiring a stronger current than normal to excite contraction, there are no qualitative changes.

On examination of the spine, no deformity could be ascertained; but there was tenderness over the mid-dorsal region. The temperature has been always normal, and physical examination revealed no organic disease of the heart, lungs or abdominal organs.

The patient still suffers from constipation, and difficulty in evacuating his bladder.

An attempt was made to relieve the pain by blisters and fomentations, and he was placed upon iodide of potassium, ten grains three times a day, on the possibility that he was suffering from compression of the cord by a gumma. He remained very much in this condition, the pain perhaps being a trifle better, till June 11th, when he suffered great pain in the hypogastrium. The water was drawn off, but after some days the urine became ammoniacal. From this time until his death, the bladder was washed out daily with one in twenty solution of boro-glyceride.

June 25th.—Morphia injections commenced on account of the pain. He is now quite unable to move the right leg, and feels tickling sensations in both limbs. It is now necessary to draw off all his water.

July 9th.—Ankle-clonus is now very marked on both sides Plantar reflexes are exaggerated, but the knee-jerk on both sides is still absent; also the abdominal and cremasteric reflexes. Anæsthesia and analgesia now at the same level on both sides. The rectum is only evacuated by enemata. He complains of a constant sense of fulness of the belly; but there is no vomiting. Pupils are normal, and the upper extremities are unaffected.

August 18th.—Patient is lying in bed on his back, complaining of a great deal of pain on the right side, which he says is greatly aggravated by twitching in his legs. For this reason he lies with his legs semi-flexed, not that he has any

power over them, but he draws them up into this position with his arms. On attempting to straighten the knee-joints, great rigidity was observable.

Ankle-clonus extremely well marked. Both feet are swollen and œdematous. The legs do not appear wasted.

The patient remained in this condition several months, daily becoming more exhausted by the pain, which was only relieved by continual hypodermic injections of morphia.

October 3rd.—Began to pass fæces unconsciously. A few days later the bedsores spread rapidly in area; and he eventually died November 15th, 1886.

Necropsy, twelve hours after death. Body emaciated; bed-

FIG. 1.—Tumour and infiltrated 5th left dorsal nerve, as seen on slitting up the dura mater. The part of the tumour in which there is no shading represents the thin part of the cyst-wall. The posterior spinal vessels run upon the surface. Natural size; somewhat diagrammatic.

sores at all the points where pressure occurred. A large sore, quite a foot across, exists over the buttocks, sacrum, and hamstrings, the muscles and bone being exposed. The skin had sloughed about the groin and the lower end of the left thigh, where pressed upon. There are also sores over the shins and heels.

Abdomen.—Superficial view normal. Liver much enlarged, weighing 6 lb. 6½ oz.; pale yellow, the edge rounded; no albuminoid change. Nothing else noteworthy in abdominal organs.

Thorax.—Pleuræ normal. R. and L. lungs healthy. Heart-substance pale; otherwise normal.

Brain—quite healthy.

Spinal Cord.—When the spinal canal was opened in the mid-dorsal region, the theca vertebralis was found swollen by a bluish mass within it. The dura mater having been slit up along the posterior surface, an oval swelling (see Fig. 1), about 1 inch long by ¼ inch thick, was found lying upon the spinal cord and the left 5th dorsal, which could be seen entering it. The tumour was cystic, and on section was found to contain a blood-stained fluid. There were large vessels, continuous with the spinal vessels, running over its surface. The nerve was swollen to three times its natural size; and was soft, red, and gelatinous, being apparently infiltrated with some new growth. It may be mentioned that a careful search of the body was made for secondary deposits, but without positive results.

A portion of the infiltrated nerve and the spinal cord were

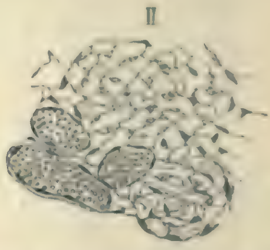


FIG. 2.—Section of a portion of the infiltrated nerve, showing branched, stellate, and spindle cells forming a reticulum with dilated vessels. Magnified, 200 diameters.

hardened in two per cent. solution of bichromate of ammonia. Sections of the former obtained by the paraffin method (Fig. 2) showed the growth to be a myxo-fibroma, which had apparently resulted from reversion of the connective tissue of the nerve to the embryonic type. It was very vascular, the vessels appearing to be dilated capillaries. The nerve was almost entirely destroyed; there were, however, a few small bundles of partially atrophied and degenerated nerve fibres left, but even here there could be seen a conversion of the connective tissue, forming the perineurion and endoneurion, into mucoid tissue. The nerve when traced up to the cord was seen to extend by the posterior root into a cyst.

Microscopical examination of the cyst—showed a fibrous capsule, embedded in which were numerous dilated vessels, suggesting the possibility that rupture of one of them had contributed to its formation. Fig. 3 shows the cord lying

beneath; and although this is completely disorganised by compression, the cyst is seen to be situated outside the pia mater, and the cord is in no way *directly* involved by the growth.

III

FIG. 3.—Transverse section of the cord and cyst, magnified 3 diameters. The vascular nature of the walls of the latter is obvious. The cord was disorganised in this situation, and the position of the grey matter is represented by a space corresponding to it in form. It will be observed that the tumour lies entirely outside the cord.

Examination of the cord, after staining by Weigert's method, exhibited the following degenerations:

1. In the *cervical* region: Posterior median, direct cerebellar, and ascending antero-lateral columns (Fig. 4, p. 216).

2. *Below* the *tumour:* crosses pyramidal tracts on both sides (Fig. 5, p. 216).

There were no changes in the multipolar cells of the anterior cornua; but apparently, from the congested state of the vessels in the posterior cornua, the grey matter in this situation might have been affected by inflammatory changes.

Comments on the Case.—There are many points of interest in this case.

1. The history points to the growth having commenced in the nerve, and having extended to the spinal canal after about four years, the patient all this time attributing his suffering to rheumatism.

2. The history and symptoms thereafter indicate a slow but increasing compression of the cord; and this led to the diagnosis of tumour. As the patient was a young man, cancer could be excluded. The diagnosis then lay between syphilitic growth, vertebral caries, tuberculous growth, some form of

sarcoma, or an uncommon non-malignant growth. *Syphilis* was improbable, because there was no history pointing in that direction, nor were the symptoms in any way relieved by anti-syphilitic treatment. *Vertebral caries.* There was no history of injury nor cause for caries, and for four years there had been little or no tenderness over the spine, everything pointing to an affection of the nerve. Moreover, very careful physical examination failed to discover any of the ordinary signs of disease of the spine.

Tuberculous tumour and sarcoma were improbable, because of the very slow course of the symptoms, and the complete

Fig. 4.—Diagram showing degenerated tracts (dotted regions) above the tumour.

Fig. 5.—Diagram showing degeneration of crossed lateral tracts below the tumour.

absence of any evidence of disease in the internal organs. Thus by a process of exclusion we might conclude that the evidence was strongly in favour of a *non-malignant growth*, spreading from the nerve or its root on to the spinal cord.

3. Tumour of a non-malignant nature having been diagnosed, we now greatly regret that an attempt was not made to remove it. On one occasion the patient expressed his willingness to submit to an operation. Had we called in a surgeon, there is but little doubt that the tumour might have been removed successfully, for it proved in no way to have invaded the cord. Mr. Victor Horsley, who has had such success with

operations on the brain, has expressed the opinion to us in conversation, that this was in every respect a case that would have been favourable for operation.[1] Dr. Gowers, in his recent work on the spinal cord, says: "It is highly probable that surgery may be able to cope, in some degree, with meningeal tumours. Modern methods render the opening of the spinal canal far less formidable than it formerly was, and the removal of a tumour from the membranes of the cord would involve less immediate danger of serious consequences than the removal of a tumour from the brain." Although myxomata of nerves are not uncommon, and those commencing in the membranes of the spinal cord not very rare, yet such a one as this, commencing in the nerve or its root and spreading on to the spinal cord, is—as far as we have been able to ascertain—unique. Notwithstanding this, we have published an account of the present case, not only from its own inherent interest, but because it appears to us to justify the belief, that instances do occasionally occur in which tumours compressing the spinal cord may be treated surgically with complete success.

4. There are some other points of interest in the case in connection with the symptoms: (1) the absence of all the reflexes except the plantar; and (2) loss of movement in the left limb, and eventually the right limb, except slight movements in the toes. Dr. Bastian, in his article on diseases of the spinal cord in Quain's "Dictionary," has noted a similar fact in a case that occurred in his practice, and he has attributed it possibly to vaso-motor spasm of the vessels in the grey matter. In our case microscopical examination showed a congested condition of the vessels of the posterior horns.

Again, the fact that sensation was more affected on the left side than the right was interesting, as well as the different levels on the right side at which the touch, temperature, and painful sensations, were lost—observations which appear to support the view that different fibres and different tracts convey these sensations upwards to the brain.

[1] He has since successfully operated in a case of spinal tumour; but the details of it have not yet been published.

A CASE OF ATAXY WITH LOSS OF MUSCULAR SENSE.

BY BYROM BRAMWELL, M.D., F.R.C.P. (EDINBURGH).

In his very interesting and important communication on "The Muscular Sense, its Nature, and Cortical Localisation," which was published in the last number of this Journal, Dr. Bastian referred to two cases, recorded in abstract by Landry, in which the "muscular sense" was lost, while the tactile sensibility of the skin was very slightly, if at all, impaired.

The importance of cases of this description, as bearing upon the subject discussed by Dr. Bastian, is so obvious, that it need not be again insisted upon; while the rarity is shown by the statements of Dr. Ferrier: "that Dr. Bastian, with all his experience, is able to adduce only two cases, both hysterical, in support of his assertion, that muscular sense may be lost apart from affection of tactile sensibility;" and again: "I have elsewhere stated that defects in muscular sense are usually associated with defects in common or tactile sensibility, and that I am not aware of any case in which there has been loss or impairment of muscular sense without coincident impairment of tactile insensibility."[1]

In the following case of locomotor ataxia, the muscular sense is profoundly impaired, while the sensibility of the skin (except inasmuch as there is hyper-sensibility to cold and to pain) is practically normal.

Locomotor Ataxia of eight years' duration; extreme inco-ordination and total abolition of the Muscular Sense in the lower extremities; tactile sensibility of the skin of the lower extremities very slightly if at all, impaired; increased sensibility to cold, and increased sensibility to pain in the same parts. Dilatation of the thoracic aorta; aortic regurgitation, hypertrophy, and dilatation of the left ventricle of the heart.

The patient, O. C., a labourer, æt. 40, is an inmate of the Comley Bank Workhouse Hospital, Edinburgh, where, through the kindness of Dr. A. H. Peddie and Dr. Manners, I have had the opportunity of investigating and of studying the case.

[1] 'BRAIN,' April, 1887, pp. 91 and 92.

Previous History.—The patient states that his illness commenced some seven or eight years ago (in 1879 or 1880), with what he terms "weakness in the abdomen" and costiveness. This "weakness" gradually increased; his walking became difficult, and he suffered from sharp, shooting- (evidently lightning-) pains in the lower extremities.

During the year 1883, he was admitted to the Edinburgh Royal Infirmary, but he received no benefit; in fact, during his stay in hospital, the condition seems to have advanced rapidly, for he was able to walk into the Infirmary, but could not walk out. He has not been able to walk since.

On two or three occasions, at the commencement of his illness, he saw double; but he is unable to remember the exact dates.

He had syphilis sixteen years ago.

Present Condition.—The patient is pale and thin, and has a worn and somewhat haggard expression of countenance. He is quite unable to stand or walk, and only seems willing or able to move the lower extremities when he sees them. If, for instance, he is made to support himself on another man's back by means of his arms placed round the bearer's neck and with his feet touching the ground, he complains bitterly that he is going to fall, manifests great emotional excitement and appearance of fear; he says he does not know where his legs are. When the other man is made to walk forward, the patient makes no effort to move his legs, which are helplessly dragged and sprawled out behind. On one occasion he was strongly urged to try and make a step or two when in this position, with the result that one of his legs was thrown forward with great force, the motor discharge and resulting muscular contraction being out of all proportion to the movement which the patient desired to accomplish; in short, he seemed absolutely ignorant of the amount of muscular force required for the performance of the muscular act.

There is, however, no paralysis in the muscles of the lower extremities. The development of the muscles in the body generally, but more especially in the lower limbs (obviously here the result of disuse) is poor; the muscles are somewhat flabby, but the muscular power of all the muscles (tested for individual movements) is, considering their size, good.

When the patient is lying on his back in bed, he can execute any desired movement with the lower extremities, so long as he can see them; and the movements which he performs in this way are fairly steady and co-ordinate. He can, for example, perform with fair precision such movements as the following:—raising the foot high into the air and then bringing it slowly down, so that the heel rests on the point of the great toe or on the knee of the opposite (passive and extended) leg.

The power of co-ordinating the muscles of the lower extremities, when the eyes are closed, and the muscular sense in the lower extremities seem quite lost.

When the eyes are closed, and he is told to execute the movements described above, the leg is wildly thrown out, first in this

direction, then in that; and there is no appearance of any intelligent attempt to execute the desired movement, all knowledge of direction and all power of co-ordination being evidently wanting. After he has attempted to make a movement, such as the above, and the leg is allowed to remain in the position which it happens to have reached, the patient is absolutely and totally ignorant of its position. If told to take hold of the foot, he gropes about for it with his hands; it may be quite in the wrong place, often, in fact, far away from its actual position, and he only finds it by mere chance or by his hand coming accidentally in contact with some part of the limb, when he feels his way (guides his hand) down the limb to the foot for which he is seeking. When the lower extremities are placed in any given position (the eyes being of course closed while the passive movement is being made), the patient is also absolutely ignorant of the position in which the foot remains after the movement has been executed. He gropes about for the foot as before, and only finds it accidentally in the manner described above. He has, however, some knowledge of the direction in which a forcible passive movement of the hip or knee is being made. He can tell, for example, whether the hip is being flexed and extended on the abdomen, or whether it is being abducted or adducted, provided that the movement of flexion and extension or of adduction and abduction, are forcibly and rapidly repeated; but he is ignorant of the direction of the movement when it is slowly and quietly performed; and he is unable, even after quick and forcible passive movement, to tell the position in which the limb remains. In the same way he can tell when the knee is being forcibly flexed and extended. He does not, however, appear able to appreciate the direction of movements at the ankle joint, even when the movements are forcibly made and quickly and rhythmically repeated.

He is quite unable to appreciate ordinary difference in weight with the lower extremities. He does not, for instance, know the difference between the lightest and heaviest of the leather-covered balls with which I am in the habit of testing the power of appreciating weights, which weigh respectively three and twenty ounces.

He is, however, able to appreciate great resistance to muscular action. If, for example, he is told to make a powerful effort to raise his leg from the bed (the eyes being closed and the foot being firmly fixed down by the hand of the physician), he is conscious that some resistance is being opposed to the movement (possibly he acquires this information to some extent, at all events, from the respiratory movements associated with a powerful effort); but he does not seem to be aware that none of the desired movements has been executed.

There seems to be no loss of muscular sense in the upper extremities. He can (with precision and accuracy) touch the tip of the nose (the eyes being of course closed) with the fore-finger of either hand; and can, in the same way, bring the tips of the two fore-fingers in accurate contact in the middle line in front of

him—a delicate test of the patient's power of appreciating the position of the parts concerned and of his power of co-ordinating the necessary movements. He can at once tell the exact position of the upper extremities after passive movements; and can accurately describe the direction of any passive movement which the upper extremities are made to perform. He can also appreciate, with the upper extremities, slight differences in weight. He correctly differentiated all the test balls.

Sensory Functions.—The shooting-pains in the lower extremities are much less severe than they were a few years ago: they seldom now trouble him unless he is exposed to cold or draughts, to which he seems particularly sensitive. He often suffers from sharp shooting-pains in the abdomen (the exact locality he was not able very definitely to define). These pains came on in paroxysms; and are sometimes so severe as to make him cry out. The abdominal pains have never been associated with vomiting or other evidence of gastric disturbance.

The tactile sensibility of the skin of the lower extremities is practically unimpaired,—the patient can feel the touch of a soft and light object (such as a young tender hawthorn leaf) on any part of the thighs and legs and over most parts of the foot. He never failed to appreciate and locate correctly over any part of the lower extremities, including the feet, the touch of a hard object, such as the fine but blunt point of a tuning-fork.

(The tactile sensibility was not tested by means of the æsthesiometer, for I have found this instrument so unsatisfactory in uneducated persons and in dealing with diseased conditions, that I have practically discarded it as a reliable and useful aid to clinical investigation.)

Round the abdomen, at the level of the umbilicus, there was certainly a band of anæsthesia—measuring two inches in vertical extent; the loss of sensibility was by no means total, in fact it consisted rather of inability to locate accurately and correctly, and in delay in appreciating tactile impressions, than of abolition of tactile sensibility. This sensory impairment was more marked on the left than on the right side.

Painful impressions were vividly felt all over both lower extremities; there was, in short, well marked hyper-algesia. There was also some hyper-sensitiveness to impressions of cold, and perhaps slight loss of sensibility to heat, but this point was very doubtful.

Reflexes.—When the soles of the feet were tickled, the patient made some complaint of pain and drew up the legs.

The knee-jerk on each side, and the plantar, cremasteric, and abdominal reflexes were entirely absent.

Urination was said to be attended with considerable difficulty. Calls to urinate come on with great suddenness, and have to be immediately obeyed (*precipitant urination* from loss of inhibitory control); much straining is required to empty bladder; and this, he says "can only be effected when he curls himself up and bends forwards," in other words, when he places himself in such a

position as to bring the full purchase of the contracting abdominal muscles to act upon the bladder.

The *bowels* are obstinately constipated.

The *pupils* are small, the right measuring 2 and the left 2½ mm.: it is doubtful if the pupils contract to light, if there is any contraction it is certainly very feeble; contraction associated with efforts, fixation for near objects and accommodation is active.

Sight, both for white and colours, seems good, and the other casual senses are normal.

Powerful pulsation can be felt in the supra-sternal notch, and there is distinct impairment of the percussion note over the manubrium-sterni. A well-marked double aortic murmur is audible over the course of the aorta and up and down the sternum. The pulse is typical of aortic regurgitation. The left ventricle is moderately enlarged, hypertrophied and dilated. The appetite is good, the tongue is clean, and the other systems and organs seem natural.

A CASE IN WHICH PARALYSIS OF THE SPHINCTERS AND INCONTINENCE OF URINE WERE, TOGETHER WITH TORPID INTELLECT, THE CHIEF SYMPTOMS OF SYMMETRICAL DISEASE OF THE CORPORA STRIATA.

BY JONATHAN HUTCHINSON, F.R.S., LL.D.

MR. S., the manager of a bank, aged 54, but looking younger, consulted me, at my own house, on account of incontinence of urine, on January 30th, 1885. The incontinence had occurred almost solely in the daytime. He said that when the desire came he could not wait, and that the urine escaped before he could get to the utensil. Yet there was no urgency of desire. Sometimes the escape would be almost without warning. This trouble had begun six weeks before, and his accidents now happened two or three times every day. I thought, of course, of enlarged prostate, but on passing a catheter found only a small quantity in the bladder, and on using the finger per anum the prostate was felt to be but slightly enlarged. In making the latter examination I realised that the sphincter ani was quite relaxed. It allowed the finger to slip in and out without gripping it in the least. The lower bowel was loaded. He denied that he had experienced incontinence of fæces, but for this the very solid condition of the fæces probably accounted. I now thought of the spine; but on enquiring, he denied that he had any weakness or alteration of sensation in his legs. He had had no headache, and asserted that his incontinence of urine was his only symptom. On looking at his pupils, I found that their margins were extensively adherent in both eyes. This much interfered with examination of their activity. He said that he had usually been a bad sleeper, but was now sleeping better. He looked well, seemed inclined to make light of his ailments, and on this occasion I saw no reason to suspect that he had any cerebral disease. I thought that he was about to become the subject of some form of spinal paralysis. On the following day I received a letter from his brother-in-

law, which showed me another phase of the case. From it I make the following extracts :—

"After our interview with you yesterday I went to Beckenham and saw my sister, and found her in great trouble about the condition into which Mr. S. seems to be rapidly falling. It appears that he *has* occasionally—certainly three times—had involuntary motions of the bowels. But her chief anxiety arises from the utter mental prostration, and physical inertia which seem to be overtaking him. This inertia is so great that he seems to be incapable of making the slightest effort. For instance, he not only cannot exert himself to get in time for his daily train, but with plenty of time to spare to catch the next, he will lose that also. And it does not seem to trouble him that this should happen frequently. Then when he comes home he will stand in an aimless way with coat and gloves on for a long time, not seeming to know or to care what he is going to do next. Mrs. S. had the greatest difficulty to get him off in time for his appointment with you. For nearly two hours, while he was dressing and getting his breakfast, she hardly dared to leave him, and it was only by having a cab waiting at the door—although the station is only five minutes' walk—that he was got off in time. Another little thing she notices is that he will sometimes grasp her hand or arm sharply enough to cause pain, or will 'flick' one of the boys with anything he is carrying, acting apparently without thought, and from some momentary impulse. In short, he is not himself at all."

A few days later, one of his colleagues in his place of business called on me and gave a similar report. He said that Mr. S., from having been an austere and precise man, had become the reverse, that he was now effusive and jocular in a manner which was quite contrary to his wont. Above all, that he was constantly passing his water as he sat in his office-chair without seeming materially annoyed about it.

On Feb. 7, a week later, I saw Mr. S. again, and ascertained the following points: that his knee-jump was good, that his pupils acted well, and that he could stand well either on his heels or on tip-toe. He told me that he had not lost sexual power, but had always been very moderate. He admitted, on being pressed, that he had occasionally a peculiar feeling in his head, which he could not explain. It was akin to giddiness, but he was not giddy. His memory and intelligence appeared to me after a long interview to be perfect, but he seemed rather too cheerful and smiled too much. It should be said that there had been no stage of elation such as occurs in most cases of the general paralysis of the insane. His sight was good, and he had no morbid changes in the fundus. He said that for ten or twelve years past the soles

of his feet had been a little numb, but they had not become more so of late. By another examination I confirmed the observation that his sphincter ani was quite lax. He repeated that he was sleeping better than he had ever done, and alleged that his head and mind were all right.

It was now arranged that he should leave town, and he went to Scotland with his wife. Here he became rapidly worse in all respects, and after three weeks they returned.

In the middle of March I saw him at his own house. He had lost flesh and strength, and looked more pale, though not markedly ill. He walked into his drawing-room to meet me, and greeted me cheerfully; but he did not seem to know what to do, and I had to ask him to sit down. He conversed about his journey, but in a vague and uninterested manner. I found that he did not know the day of the week, but after I had told him, he corrected me as to the day of the month. All other symptoms were as before. His wife said that she was obliged to make him get out of bed three or four times in the night, or he would certainly pass water without caring to rise. Once when on a visit to a friend, he had passed water in the room, and seemed quite careless about it. I made him whistle, and ascertained that he had no paralysis of the seventh nerve, though it was perhaps feeble. Within a week of this visit Mr. S. died. I had not seen him again, but I obtained the following facts from his surgeon and his wife. During the week he got a little worse, but he had seemed decidedly better on the day preceding his death. He had been up every day, and about the house as usual. At length, one morning after a night of unusually heavy sleep, he had been difficult to wake, and when roused and asked if he would get up, had replied distinctly, "No." His wife kissed him, and he kissed her back, but then immediately sank into sleep again. The sleep became more profound, and they could not rouse him. His left arm and his right leg were engaged in twitching convulsive movements. The sleep passed into coma, which was attended by stertor. Then the respirations became intermittent, and finally they ceased. He died six hours after he fell asleep.

We got permission to make a post-mortem. It was done, under considerable difficulties, about thirty-six hours after death. In the inner side of the left corpus striatum was a soft pink semi-gelatinous growth. This growth bulged into the ventricle, and was quite smooth on its inner surface. On its outer side it merged without any boundary line into the substance of the corpus. The thickness of the new growth was about half an inch. A little to its outer side, in the white substance of the hemisphere, were two small islands of

similar structure, as large as cherry-stones, but quite ill-defined. Neither these nor the larger growth had any softening or congestion around them. At this level the opposite hemisphere appeared healthy, but on slicing it more superficially, there was found just above the corpus, or in its upper part, a large softened area broken down by blood clot, but having, at one part, an indistinct lining membrane. A clot the size of a walnut, soft and jelly-like, extended from this patch of disease into the ventricle.

There was not a trace of disease at the base of the brain, nor in the pons or medulla. The arachnoid over the surface of the hemispheres on both sides was adherent, but there was nothing very unusual or which appeared to be recent.

The examination of the brain was not quite so precise as could have been wished, but it was done by candle-light, and under disadvantages. It may be taken as established that there was destructive disease of the anterior and inner parts of the corpus striatum on both sides, and that no inflammatory mischief had been set up. The hæmorrhage was on the right side only. No doubt this bleeding afforded the explanation of the suddenly fatal event.

My eldest son made a careful microscopic examination of the tumour, and has preserved sections. I append his report.

"After hardening with the usual reagents, some satisfactory sections were obtained, fully bearing out the diagnosis of sarcoma made at the post-mortem examination. Those parts free from blood-infiltration showed spindle and round cells closely massed together, and tending to be arranged somewhat in lines. From the size and shape of the cells, the tumour should perhaps be regarded rather as a mixed round and spindle-celled sarcoma, than as a typical glioma, though presumably its origin was from the neuroglia.

"Especially in the neighbourhood of the hæmorrhage some vessels of fair size were seen, with a considerable number of capillaries, probably of new formation, in which the thick cellular wall almost concealed the lumen. Even in some parts which to the naked eye looked like blood-clot, tumour elements could here and there be made out."

It is certainly remarkable that a lesion in this position should have been attended by so much mental disorder and by no limb paralysis. Sphincter-paralysis had been the most conspicuous symptom thoughout, but his limbs, so far as he would admit, had never failed. He could always grasp firmly with his hands, and could walk. His walking power had, however, certainly diminished. It is very possible that the symmetry of the disease had been productive of bilateral weakness, and that thus the defect had been less easy of discovery.

After his death, his wife told me that she had sometimes thought that his right limbs were weak, but one of his sons who had been much with him had formed the same impression as to his left. It was certain, they said, that in walking he sometimes reeled a little to one or the other side.

Dr. Bright, in the second volume of his "Medical Reports," has given a portrait, which shows a closely similar condition of the corpus striatum. The chief differences indeed, between his case and mine consists in the fact, that in his the lesion was on one side only, whilst in mine it was symmetrical. If we keep this fact in mind, we shall find that the symptoms in the two cases were nearly parallel. In Dr. Bright's case, as in mine, incontinence of urine was an early symptom, and throughout one of the most conspicuous. His patient was a man of 40, who had been a soldier. The earliest symptoms observed had been that he would occasionally fall in the street, and lose consciousness for a moment or two. He complained of pain in the forehead, and had occasional absence of mind with slight weakness of the left side. Next he began to constantly pass urine in bed, and occasionally fæces also. After this there was general muscular and mental debility, with strabismus and diplopia. "His manner was peculiar, and his conversation incoherent; he stood for some minutes together in one position as if lost in abstraction; when spoken to, he answered a question with some consideration apparently, but then at once ran on to something else, without, however, saying much, but making some statement perfectly without foundation." The symptoms, including the premonitory group, had lasted a year when Dr. Bright first saw him, and he lived, afterwards, in Guy's Hospital, for six months longer. He had no facial paralysis, nor, so far as the notes inform us, any defect of sight or hearing. A few days before death it is noted: "His general manner and aspect quite unchanged. He stands about musing, and if you ask him how he finds himself, answers after a few seconds of consideration, 'I am not quite dead.'" He had gradually become more dull and heavy. It was only during the last week that his imbecility and debility were such that he could not leave his bed. He had on the last day great difficulty in swallowing.

Dr. Bright does not describe the condition of the corpus striatum as implying new growth, but from the appearances delineated, it probably did so. He says that two-thirds of corpus were "involved in softening, and presented a filamentary and watery-brown appearance." The rest of the brain-substance was healthy, but over a space two inches square on the right hemisphere all the membranes adhered firmly. There was some general serous effusion. In commenting on the case,

Dr. Bright states: "The appearances after death were confined to softening of the corpus striatum on one side . . . but it had not the precise appearance of an apoplectic clot."

In Auvert's magnificent collection of pathological plates there is one (Pl. 68, Fig. I.) which shows the section of a brain in which appearances are represented somewhat similar to those I have described. The tumour involved both thalamus opticus and corpus striatum. The patient, a man of 56, had died after a week's illness, following a sudden seizure during anger. He had been liable to hemicrania. The disease is described as red softening, and not as new growth.

CASE OF THOMSEN'S DISEASE.[1]

BY FRENCH BANHAM, M.A., M.D.

Physician to the Royal Berkshire Hospital.

THE patient, a brief report of whose case is subjoined, came under my notice a year and a half since. I am now again in communication with him, and as the disease from which he suffers is one of great rarity, I feel that so good an example of it should not pass unrecorded.

The affection of which he is the subject is known as Thomsen's Disease (so named from the physician who first described it, and who is himself a sufferer from it), and which consists almost wholly in spasms of the voluntary muscles coming on at the outset, or upon any change of motion, and quickly passing off again. Few, if any cases, have been recorded in our English journals, so in pointing out any special peculiarities exhibited by my patient, I take as my standard of comparison the descriptions of the disease as detailed in some of our best treatises on Diseases of the Nervous System. The affection is generally spoken of as being hereditary; but in this case it seems not to be so, as no other member of the family, as far as can be ascertained, has been similarly affected, nor is there any tendency to disorders of the nervous system, and the patient is one of seven, the eldest being 22, and the youngest 9 years of age. Further, it is said to be congenital, some indications of it having been observed in the cradle; but my patient, now 18 years of age, had no symptoms until he was 12, and these did not become severe until two years ago, when he was taking swimming lessons, and for forty consecutive days was accustomed to remain in a cold bath for upwards of an hour together. He is an intelligent youth, well-developed, especially as regards the legs, and muscularly strong, while his general health is good.

The first time he noticed a spasm was, as I have said, six years ago. He was jumping over a flower-bed in his schoolmaster's garden when a spasm of the legs occurred; he could not lift his feet so as to clear the flowers, and his master

[1] Read before the Reading Pathological Society, Dec. 8th, 1886.

caned him for the damage done. He remembers the increasing frequency of the symptoms from that time, marked by his inability to join in such games as "Hare and Hounds," as he had formerly done.

The symptoms vary very greatly at different times. When severe, a spasm comes on as soon as he has altered the condition of a muscle after a period of rest. Thus, after having sat down for a little time, when he rises to his feet, he cannot stir his legs because the spasm immediately occurs, and for the same reason he cannot balance himself unless he has gained his equilibrium before the spasm is developed. This leads to his being thrown down in trains and tramcars on attempting to stand up after sitting, unless he is able first to grasp the rail. Again, after getting into a conveyance and sitting down, he cannot bring his heels towards the seat at once, but often kicks the opposite passenger through his legs sticking out stiffly; and as an illustration of the transient character of some of the spasms, I will quote his own remark—"that he generally gets his legs down before the fellow he has kicked begins to swear." The spasms are also very marked in his attempt to walk upstairs. He has very great difficulty in lifting each leg on to the first and second steps respectively. A slightly less difficulty in the next two series of motions, and a continually decreasing difficulty in each successive series, until he has mounted, say, twelve or fourteen steps. After this he has no further trouble for the time being. In such circumstances, twelve or fourteen seconds is the longest time that the difficulty lasts. Again, on bending the arm sharply at the elbow, the hand may readily approach the shoulder, and will return about half-way back, when a spasm occurs which holds it there for a few seconds. Again, on opening the mouth, it is fixed for a few moments before he can close it. The difficulty vanishes, however, after a little exercise, and will not recur unless brought on by fright or great excitement. His arm or jaw seems never to remain rigid for longer than about three seconds; the time, however, varies from this to a momentary check in the movements. When a muscle has become stiff, as he expresses it, he does not suddenly acquire full power over it again, but in a longer or shorter time he can begin to move the limb, slowly at first, but with increasing vigour as time passes.

The disorder varies in intensity, and when less severe, the spasms do not take place so immediately at the outset, but only after a series of motions. For example—he will mount nine steps readily when a short spasm will occur, lasting perhaps until he has reached the twelfth, and necessitating his grasping the banister to prevent himself falling.

There are certain circumstances and states of mind which seem to influence the spasms. They are slightly more marked, and last longer when he directs his attention to them; but his will does not seem to affect them at all. Under fear or excitement, the symptoms will suddenly appear at other times than at the outset of voluntary motion. As an illustration, I may mention that when walking one day with a friend in a railway tunnel, and being surprised by the near approach of a train, the spasms suddenly set in in the legs, so that his companion had actually to drag him out of the train's way. And again, sometimes on getting into a train he has literally to fall into the carriage if he is greatly excited or the train is on the point of starting. In the case of the hands, the spasms so affect them that he is frequently unable at once to let go objects that he has grasped. The muscles of the tongue and larynx, so far as I can ascertain, have never been affected. He seems always to have led an active life, so that I am unable to say what the effect of rest would be upon the tendency to spasm. It seems, however, that when after remaining in one position subsequently to taking prolonged exercise, he recommences movement, the spasms at the outset occur very promptly and strongly. The disease is considerably influenced by cold, rainy weather, and he tells me that he generally knows when it is going to rain by the strength and character of the spasms. The tendon-reflexes are scarcely so strong as is usual in health, and the left patellar tendon-reflex is rather weaker than the right. In regard to the superficial reflexes, tickling of the soles of the feet elicited no response.

The electrical reactions of the muscles are not precisely coincident with what has been recorded in other cases. Momentary stimulation of the nerves did not cause more than a momentary contraction of the muscles either in the case of the opening or closing contractions of either pole, or in the case of a momentary stimulation with a rapidly intermittent current. The contractions produced on starting a current through a motor point were momentary and normal, although the current was maintained constant after making. I mention this because it has been stated that in some cases in certain muscles an unbroken current has caused peculiar wave-like contractions, about one per second passing from the negative to the positive pole. An intermittent direct current, however, that is an intermittent current of uniform direction, maintained a constant contraction of the muscle with either pole. In the case of one muscle only, the vastus internus, peculiar contractions and relaxations were observed during the application of the intermittent current, the active electrode being

placed over the motor point of the muscle. At first a normal contraction lasting about two seconds occurred, and then an irregular recurring series of contractions and relaxations. In all the experiments the sequence of the polar reactions was found to be normal, with the slight exception that the ACC and the AOC were produced together. The first contraction was produced with a kathodal current of 6 milliampères, the diameter of the electrode being 1 inch, the ACC and the AOC with a current of 8 milliampères, although the opening contracture was more vigorous than that produced by the closing of the anodal current with the same current strength. The KOC did not occur until the current had been increased to 10 milliampères.

I have not made any microscopical examination of the muscular fibre; but in a case observed by Erb many fibres were seen exceeding the normal maximum in size.

I must thank Mr. J. Lester Taylor, of Sheffield, for the kind assistance he has given me in working out some of the details of this case.

I.—A CASE OF RECURRING ATTACKS OF TRANSIENT APHASIA AND RIGHT HEMIPLEGIA.

II.—A CASE OF TUMOUR OF THE PINEAL GLAND.

BY EDWARD O. DALY, M.A., M.D. (OXON.), M.R.C.P.,

Physician to the Hull Royal Infirmary.

A GENTLEMAN, aged 68, was suddenly seized, on March 11th, at 6 A.M., with aphasia and right hemiplegia. The attack lasted about five minutes, and passed off as rapidly as it came on. He had suffered occasionally for years from gout, most frequently affecting his bladder. The first sound of his heart was found to be feeble; his arteries were thickened, and his urine was pale with a well-marked trace of albumen, and a sp. gr. of 1010. His temperature was normal; his pulse 80. On March 13th he had two attacks of a precisely similar kind to the first, each lasting about half an hour.

March 14th there were ten attacks, the shortest lasting about ten minutes, the longest almost an hour.

March 15th there was one attack of ten minutes' duration, and three in succession, with about a ten minutes' interval between, each extending over three hours.

Since March 15th he has been quite free from an attack of any kind.

I saw this gentleman during several of these attacks. They came on perfectly suddenly without any premonitory symptoms, and passed off as rapidly. He would suddenly say, "I am all right again."

There was never any kind of convulsion.

During the attack the intelligence was certainly affected, but it was difficult to estimate to what extent.

When asked to put his tongue out he did so at once, and also closed his eyes, and performed intelligently all actions asked of him. The aphasia was not complete, but the words he had at his command were limited, and frequently misapplied; and in some of the attacks he was quite unintelligible.

The hemiplegia was also partial, more marked in the arm than the leg. He could move the right hand to a limited

extent, and was able to grasp my hand feebly. There was also slight anaesthesia of the right side.

The attacks in every instance ended abruptly, the speech becoming instantaneously perfect. The power in his right side returned almost as rapidly, but after the longer attacks a few minutes elapsed after the return of speech before he could use his arm and leg perfectly.

In the intervals between the attacks he felt quite well, with the exception of slight frontal headache.

The only treatment consisted of perfect rest, a few grains of calomel, and a mixture containing small doses of iodide of potassium and citrate of lithia.

When I first saw this case I was inclined to think it due to transient thrombosis of the left sylvian artery. On referring to various books on the nervous system I find Dr. Bastian, in his book 'Paralysis from Brain Disease,' mentions spasm of the vessels as one of the causes capable of rapidly producing a hemiplegic condition. He remarks unfortunately there is a great deal of uncertainty on this point.

As these attacks came on and passed off so rapidly, the speech and movements of the right side in the interval between the attacks being perfect, the case seems more readily explainable as due to spasm of the sylvian artery, than to thrombosis or any other cause. The case, at any rate, seems a very unusual one.

In some cases polyphagia has been ascribed to pressure of the tumour on the "diabetic centre," but no such explanation is admissible in this case, the urine being normal throughout.

In other cases it depends on the implication of the cerebral centre of the stomachic branches of the vagus.

Again, unnaturally large appetites have been observed in disease of the post-cerebral lobes, which must have been subjected to abnormal pressure in the present case. It may be that this symptom has been noted in other cases of tumour of the pineal gland, though I am not aware of it.

This gland, now shown to be a rudimentary "parietal eye," was at one time believed to be the seat of the soul, and had the patient lived and died in those days, his moral and physical deterioration would doubtless have been brought forward as strong evidence in favour of the correctness of this theory.

Case II.—W. O., single, a labourer, aged 23, was admitted into the infirmary on October 30th, 1885. There was no family history of consumption, nor of any other constitutional disease. He had not had syphilis, and there was no history of any former head injury. He was strong and healthy until two or three weeks prior to admission, when he believed he had been

struck by lightning, and had complained since that time of more or less constant headache and photophobia. On admission he was found to be a strongly-built man, his gait was peculiar, and resembled the walk of a drunken man, swinging his trunk from side to side and from back to front. His gait was worse when he closed his eyes. As he lay in bed, he could move his legs freely in any direction. His arms were not affected, and the patellar tendon reflex was normal.

The patient complained of pain at the back of his head. His vision was impaired, pupils dilated and equal, contracting but slightly to light or accommodation. An ophthalmoscopic examination revealed double optic neuritis with swollen disc. His other senses were normal. He was constantly sick, irrespectively of food. His urine was free from albumen and sugar, and not increased in amount. Nov. 11th, slight paralysis of the left external rectus was noticed, and on Nov. 20th facial paralysis on the right side. Nov. 25th, there was some difficulty in articulation for some hours, which passed off entirely.

During the latter part of November and December attacks of spasmodic contractions of the muscles of the limbs and trunk occurred from time to time. At times his mind seemed much affected: he would get out of bed several times during the night, and occasionally wanted to fight. From the beginning of January, 1886, he began to develop a very large appetite, and his vomiting considerably abated.

He remained *in statu quo* for some weeks, and was discharged, at his request, January 29th, 1886. Re-admitted February 27th, 1886.

It was now noticed for the first time that the hearing of both ears was defective, and he was found to be almost blind. He was said to have had occasional fits. He could only walk and stand with assistance, though he could move his arms and legs freely. There was no anæsthesia. His appetite was soon found to be enormous, and the patients were in the habit of giving him what they left on their plates. On one occasion eight or nine patients put their pudding at his disposal, in order to see how much he would take; all this he finished without trouble. During the time he was in the infirmary he put on nearly five stone. The urine remained normal in all respects. During March and April he seemed to lose all intelligence, became almost completely deaf, and his sight failure passed into total blindness. He also completely lost control over the bladder and rectum. When pinched or moved he would give a kind of grunt, but hardly ever uttered a sound, except when he wanted food. He was occasionally observed to lie with his head drawn back, which was found to depend on a tonic contraction of the muscles of

the back of the neck. He had from time to time epileptiform convulsions, in which the movements were said by the nurse to have been general. I never however myself saw him in one of these attacks after his re-admission.

During the last week or ten days of his life his pulse and respiration became very rapid, and he passed into a state of coma several days before his death, on May 19th, 1886.

At the post-mortem examination a circumscribed tumour was discovered extending backwards from the point which corresponded to the situation of the pineal gland. It was loosely attached to the upper surface of the cerebellum, lying between it and the under surface of the cerebrum. In its growth it had seriously pressed upon the neighbouring structures, especially the corpora quadrigemina, which were soft and flattened. The tumour was two inches in length, one and three-quarters in breadth, and half an inch in thickness; and is well shown in the photograph kindly taken for me by Mr. Howlett. Subsequent microscopic examination showed the tumour to be one of alveolar cancer: this view of its minute structure was corroborated by Mr. Young, of Manchester.

Remarks.—The case was diagnosed with considerable confidence as a tumour of the middle lobe of the cerebellum. The attacks of spasm observed have been usually ascribed to tumours in that situation, and this growth no doubt must have pressed upon the middle lobe. I have lately however seen a case in which rigidity and tetanus-like seizures were absent from first to last, but in which at the post-mortem the middle lobe was almost entirely destroyed by a soft growth. I am therefore inclined to agree with the explanation put forward by Dr. Sharkey in his Gulstonian lectures last year; namely, that the symptoms in this case depend, not upon the pressure on the middle lobe of the cerebellum, but upon the fact, that the tumour was in such a position and of such a size as to cause pressure on the pons and medulla.

The great impairment of vision present at such an early stage of the disease was doubtless due to the fact, that the tumour from the first pressed upon the corpora quadrigemina.

Although the tumour was diagnosed during life as cerebellar, there was one symptom, polyphagia, which is not, I think, characteristic of tumour in that situation. That central disease may give rise to ravenous appetite is frequently well exemplified in certain forms of insanity. When it occurs in the case of cerebral tumours it does not, as a rule, prevent the progressive emaciation which is so commonly observed towards the close of such cases. In the present case however the patient put on weight up to the last, and was five stone heavier at his death than on his admission.

TWO CASES OF PECULIAR MOVEMENTS IN CHILDREN.

BY W. HALE WHITE, M.D.,

Senior Assistant Physician to Guy's Hospital.

CASE I.—A. A., æt. 14, came to my Out-patients' department on March 2nd, 1886. The notes made at the time are as follows:—

He is healthy-looking and very intelligent, being at the top standard but one at the Board School. He is an only child; a paternal uncle is in an asylum, otherwise the family history is very good. Forceps were used at birth, from which time his mother has noticed irregular twitchings of his muscles. When he was three months old, she took him to the Great Ormond Street Hospital. The doctor said he would grow out of it, and gave him cod-liver oil; but he has not improved.

Present Condition.—On watching the boy, occasional slow involuntary movements of the fingers and forearms are seen more on the right side than the left, where indeed they are hardly perceptible. These movements affect the face and both extremities on the slightest attempt at voluntary movement, and indeed they may at times be seen on the face without any voluntary motion. They are least marked in the lower extremity. When at their greatest they are extreme, the platysma and trapezius showing the irregular spasmodic contractions very well. It cannot be made out that the muscles supplied by the 3rd or 5th nerves are affected. Perhaps the action which shows the trouble to the greatest perfection is unbuttoning his shirt: he makes the most irregular and violent attempts with his right hand; the right side of the face is distorted; the left hand tries to help, but is affected in the same way but to a less degree, and finally his mother has to help him; but if he employ only the left hand he can after some time manage it. His handwriting is hardly legible, and he has to hold the right hand, which holds the pen, steady with the left. Owing to the face-affection the speech is irregular and jerky. The abdominal and thoracic muscles are not affected, but the affection of the right leg causes an irregular gait. All his troubles are worse when his attention is directed to them. The knee-jerks are normal, as is the triceps on the left side; but the attempt to get it on the right sets up the irregular movements. There is ankle-clonus on both sides, and diminished superficial reflexes all over the body. No paralysis or contraction, no atrophy, no paradoxical contraction. Electrical reactions all normal. No disturbances of

sensation. Ophthalmoscopic and laryngeal examination showed normal results. Special senses normal; no trophic changes. No asymmetry of head, limbs, &c.; no wasting; throat normal; uvule bifid; dentition is said to have been delayed. There is a narrow patch on the right leg.

March 1887.—For the past year he has been treated with iodide of potassium and tonics, but without any benefit. A most thorough examination showed the condition to be unchanged, except that no ankle-clonus could be obtained, and the reflexes were not so lively. He has not suffered from headache or sickness. As he has got used to me he does not make quite such extreme movements as he did at first when talking to me, but he is just as bad as ever with strangers. This patient was exhibited at a Clinical Meeting of the Neurological Society last March.

CASE II.—C. L., æt. 10, admitted under me into Philip Ward, Feb. 26th, 1887, for spasmodic movements of the right arm and right facial paralysis.

Family History.—The maternal great-grandfather had fits; he died, æt. 60, paralysed. A maternal great-aunt had fits; a first cousin (mother's sister's child) has fits and is weak mentally. The patient is the eldest of four. The second, a boy, was born with a large head and is very excitable. The third, a boy, has often been operated on for nævi. The fourth, a girl, is healthy. The mother is healthy, but her mother from childhood has had tremblings of the head and right arm: she has had thirteen or fourteen children, and is now almost bedridden.

Personal History.—His birth was a "hard confinement;" he was a nervous child, easily frightened, screaming when the clock struck. In May, 1886, he woke up one morning feeling sick and feverish. He was kept in bed a week, as he had a sore throat; his mother noticed he was a little shaky, both hands being tremulous. He quite recovered. In July, 1886, his mother again noticed tremulousness, confined to the right hand, though he could still feed himself; but by October it had got so bad that he could not write. At this time he described a pain which began in the right mid-finger and extended up the arm. He was now removed from school, where he had been looked upon as a dull boy, although he was in the second standard. His speech was noticed to be affected. By November the right arm was useless. He was taken to a doctor, who drew attention to slight facial paralysis and dragging of the right leg, and said he had either chorea or paralysis. He was taken to a hospital, where the same diagnosis was made. Change of air was frequently tried, but he got no better, and was admitted into Guy's. No rheumatism.

Condition on Admission.—Well-nourished, healthy-looking, dark hair and eyes; sits up in bed, amusing himself with toys and using the left hand exclusively. Has a small nævoid patch on the left outer malleolus. There is nothing noteworthy about the mental condition. As far as the facial paralysis permits, the pantomimic speech is good, as is the spoken, except for slow drawling but

distinct utterance; this also would appear to be due to his paralysis. There is no aphasia. He cannot write. The only defects of the eyes are that the left pupil reacts better than the right; also, on looking up, the right eye is turned further up than the left; and in moving up, it performs slight horizontal oscillations. Hearing, taste, and smell normal.

Motor Functions.—There is paralysis of the muscles supplied by the right facial nerve, but it is very variable, the mouth being drawn violently to the left at one time, but not a few minutes after; both orbiculares palpebrarum and occipito-frontales muscles act equally and well. There is some paralysis of the left levator palpebræ. The tongue is protruded normally. The uvula and palatine arches are equal. The power in both arms is diminished, but especially in the right. There is no paresis of the legs. When he sits naturally, the following is the position of the right upper extremity. The humerus is at the side, the forearm at a right angle over the chest, the hand is clenched, the thumb being adducted and pressed into the palm by the fingers. At irregular intervals spasmodic twitchings come on, mostly affecting the elbow joint, the closed hand being thus rubbed up and down the sternum and making an excursion of about two inches. About a dozen of these twitches occur in as many seconds, and then the arm is still again for some minutes. If he is being watched, the movements are continuous; and if he is told to take one's hand, the whole extremity is irregularly thrown about before the object is accomplished. All this time the hand continues flexed, and he has to open it with the right before he can grasp anything. Occasionally the hand will open spontaneously. Although there is generally much rigidity at the elbow and hand, still if they are opened with some force they soon become flaccid, and also they may at times be opened with less trouble than at others; but the flaccidity soon gives way to contraction again, and the hand shuts up. The left arm is normal, except that at first there appeared to be some slight choreiform movements of it; but after he had been in a day or two, they disappeared. No wasting, anæsthesia, or reflexes of arm. In the right lower extremity the knee-jerk is absent, as is ankle-clonus and paradoxical contraction. As he lies in bed the foot is persistently extended at the ankle, the sole is arched, the toes are extended at the metatarso-phalangeal joint and flexed at the phalangeal; plantar reflex very sluggish. On walking, this rigidity of the foot passes off, but the patient has some inco-ordination, the right leg being swung too far round, and he very quickly throws the weight on to the left. Right lower normal. No electrical examination permitted.

Cases like these in some particulars have been recorded by Ross[1] and by Hadden.[2] Most of the cases by Ross had more spastic rigidity than either of mine; the last is perhaps

[1] 'BRAIN,' Vol. V. p. 344.
[2] 'Clin. Soc. Trans.,' vol. xviii. p. 221.

doubtfully congenital, although it may be looked upon as a congenital disease supervening late in childhood; the first certainly is congenital. It is evident, on reading the accounts of them, that there is great difficulty in assigning to any one lesion the production of the symptoms in either case.

Until we know whether a lesion is present, and if so, what its position is, we have hardly enough basis for speculation as to causation of the symptoms. I intend to keep an eye on these two patients, and, if I outlive them, to complete the account of them. The cases that have been published of congenital brain deficiency in children producing symptoms, differ so among themselves, that they hardly form any guide in other cases. It is only by the recording of all these vague cases in which a diagnosis cannot be made that we can hope to eventually classify them, when we have had the opportunity of comparing a large number.

MIGRAINE ATTACKS FOLLOWED BY TEMPORARY PARALYSIS OF THE THIRD NERVE.

BY C. W. SUCKLING, M.D. (LOND.), M.R.C.P.,

Physician to the Queen's Hospital, Birmingham.

R. R., a youth aged 18, a tool maker, applied at the Birmingham Eye Hospital for treatment for dropping of the left eyelid. Dr. White found that there was complete paralysis of the left third nerve, and there being a history of previous similar attacks, he kindly sent the case, as one of interest, to me.

Since infancy the patient has been subject to severe headaches, occurring at varying intervals. During the last four years the attacks have occurred more frequently and have been more severe, and for the past two years he has had an attack pretty regularly every fortnight.

The patient's aunt used to suffer severely from periodical headache; but neither of his parents have been affected by this or any other neurosis.

An attack lasts two days, and takes the following course:—He first feels a slight pain above the left brow, which gradually increases in intensity, and is followed by a flow of saliva into the mouth, and he feels very cold. He at once goes to bed on feeling the onset of an attack, but cannot sleep, and he has to remain in bed for two days without being able to sleep or to take any food.

The pain increases gradually, and attains its maximum in twenty-four hours: it then as gradually subsides, and ends completely in another twenty-four hours.

The pain remains localised over the left brow; he does not vomit, and there are no ocular spectra or paræsthesiæ of any kind, except that he complains of a nasty taste in the mouth.

After an attack his scalp is tender, and he cannot comb his hair.

The left eyelid was first noticed to be drooped after an attack in his infancy, and this has occurred very frequently since.

Three years ago, after a severe attack, his left eye was completely closed, and the eyeball turned outward.

The eye is not affected in every attack, but only in the severe ones; and the more severe the pain, the greater is the amount of dropping of the lid which follows.

The left eyelid begins to drop at the end of an attack when the pain is wearing off. The lid takes twenty-four hours to drop completely, and in another twenty-four hours has usually recovered. The eye is not always turned outwards, but it has been so on two separate occasions, and then the paralysis lasts longer, not recovering for a week after the attack.

His mother attributes the increase in the severity and frequency of the attacks to his having attended a night school during the past two years.

On examination a week after an attack the left pupil was a little larger than the right, and responded normally to light and to accommodative efforts. The left palpebral fissure was a little smaller than the right, and the upward movement of the left eye was defective, there being diplopia in looking upwards. He complains that the sight of the left eye is not so good as that of the right. Dr. White reports that vision is normal, and that when he saw the boy the paralysis was complete both of the internal and external muscles of the eye.

The salivation that occurs in this case during the attack is also of interest.

In another case of migraine at present under my care a profuse clear discharge runs from the left nostril during the attack—the pain being located over the left brow; and in this case the scalp is tender, and painful lumps are occasionally present on it.

Migraine in some cases is so serious a malady that it completely incapacitates its victims for their duties, and in the majority of cases is a most serious drawback. Its victims can usually tell what are the exciting causes, and affirm that fatigue, worry, reading, cold winds, damp, wines, pastry, &c., will bring on an attack; but however careful they may be, they cannot rid themselves of their burden. Cigar-smoking is a very powerful exciting cause of an attack. A medical friend of mine invariably suffers from a severe attack if he smokes cigars.

In the above-mentioned case guarana in thirty-grain doses, taken every hour at the commencement of the attack, has given great relief. The duration of the attack is shortened to a few hours, and though the patient has had attacks, they have not again been followed by the paralysis.

The only other case of migraine in which the third nerve has been paralysed that I can find recorded is that published by Dr. Saundby in the 'Lancet' of January 10th, 1885.

Critical Digests.

RECENT OBSERVATIONS ON PROGRESSIVE MUSCULAR ATROPHY.

BY H. H. TOOTH, M.D.

THE great impulse given to the study of the disease, or rather group of diseases, known as Progressive Muscular Atrophy, by the researches of Erb, Charcot, Landouzy and Déjérine, Schultze and others, renders a review of the subject a desideratum. Such a review to date appeared in 1885, from the able pen of M. Charcot, but since then further advances have been made. The object of the present article is rather to place before the reader the most recent views of authors on the subject, than to criticise those views. It will be seen that recent anatomical observations tend rather to upset our preconceived ideas as to the causation of the disease. For instance, we have been taught to regard the existence of atrophy, commencing in the small hand-muscles, accompanied by fibrillar tremors, and late reaction of degeneration in affected muscles, as pointing certainly to some lesion of the cells in the grey matter of the anterior cornua. On the other hand, in an undoubted myopathy, such as pseudo-hypertrophic muscular paralysis, so called, we have never expected to find either fibrillar tremor or reaction of degeneration; and it is on those signs that diagnosis has hitherto been largely based. As will be seen, when studying Schultze's latest observation, we can no longer, apparently, rely with certainty upon these signs, and unfortunately we have not yet any to take their place.

There have always been authors who have maintained that all cases of idiopathic muscular atrophy are due to primary disease of the muscles, the nervous changes being, according to them, secondary. Such is the opinion of Friedreich, Schultze, Lichtheim, Liebermeister, and others. A fatal objection to the universal adoption of this theory is the fact, that we may have the most advanced atrophy of the muscles, with an absolutely normal condition of the nervous system.

It would appear to the impartial student that there may exist, under the comprehensive head of progressive muscular atrophy, a number of forms having the common characteristic of atrophy, but due to various causes. Thus we may have atrophies of *myelopathic* origin, due to disease of the anterior horns of the

spinal grey matter; or we may have *myopathic* atrophies, in which the seat of disease lies *solely* in the muscles themselves; or lastly, we may have *neuropathic* atrophies, in which we must look for the lesion in the *nerves* themselves. The cases hitherto recorded may most of them be classed under these three heads, but it must be admitted that there are still many anomalous cases.

There has not been added much that is new concerning myelopathies; but the recognition of the two other groups above mentioned has had the effect of considerably limiting their number, and at the same time of making their symptomatology more sharply defined. Thus the *thenar form* has become the type of a myelopathic atrophy, and though apparently in some true myelopathies the atrophy may commence in other than the hand-muscles, the number of such atypical cases is becoming less and less. The commencement of the disease in the small hand-muscles, at first sight so difficult to understand, has had great light thrown upon it by the work of Ferrier and Yeo[1] on monkeys, by which it is shown that the small hand-muscles are connected with centres in the dorsal cord at about the level of the second dorsal roots. Beevor[2] has illustrated the clinical bearing of this in myelopathies. From these facts we may speak of the thenar type of progressive muscular atrophy as a poliomyelitis, attacking first the anterior grey cells in the region of the upper dorsal roots and spreading upwards and downwards from that focus. It is, however, highly probable that a simple protopathic lesion of the grey cells, uncomplicated by any affection of the white matter, is much rarer than is usually supposed. This is Schultze's[3] opinion, and he goes so far as to assert that only two such autopsies are as yet recorded, namely those of Charcot and Gombault,[4] and Pierret and Troisier.[5] It is a fact that in many recorded autopsies there is coincident affection of the white matter.

It is in the class of atrophies in which the seat of the disease lies wholly in the muscles themselves, that the most important advances have been made. One of the earliest recognised myopathies is pseudo-hypertrophic muscular paralysis. It is needless to dwell on the symptomatology of a disease so well known, but it will not be out of place to mention the grounds on which the diagnosis of a myopathy from a myelopathy has hitherto been based.

As a rule, *fibrillar tremors* are not present in myopathies, it is true that in Schultze's[6] case, to be mentioned more fully later on

[1] Ferrier and Yeo, 'Proc. Roy. Soc.,' 1881.
[2] Beevor, 'Med. Chir. Trans.,' 1885.
[3] Schultze, 'Ueber den mit Hypertrophie verbundenen Progressiven Muskelschwund,' etc.: Wiesbaden, 1886.
[4] Charcot et Gombault, referred to by Charcot in 'Maladies du Système nerveux,' t. ii., p. 206, 4e éd.
[5] Pierret et Troisier, "Note sur deux cas d'atroph. musc. progr."—'Arch. de Phys. norm. et path.,' 1875, p. 236.
[6] Op. cit.

they were noted at an early period of the disease; but this is very exceptional, and when these little tremors are present, we may, with tolerable certainty, exclude myopathy pure and simple.

Till quite lately, it was believed that reaction of degeneration in the atrophic muscles was a sign of some nervous lesion, central or peripheral, and that it was never to be found in simple affection of the muscles. This, however, can no longer be relied on, for Zimmerlin, Schultze, Landouzy and Déjérine have shown that it may be present in pure myopathies. *Hypertrophy* of certain muscles is very often present in myopathies, but has never been seen in myelopathies or neuropathies. Very important points in forming a diagnosis are, the age at onset, the point of attack, the existence or non-existence of heredity, which is very common in myopathies and neuropathies, but not in myelopathies.

A myopathy nearly allied to pseudo-hypertrophic paralysis is the so-called "Juvenile Form," first recognised by Erb in 1884.[1] This form seems to have obtained general acceptance as a distinct type. It differs from pseudo-hypertrophic paralysis in the fact that hypertrophy is a very subordinate feature, while atrophy is a prominent one; in fact, hypertrophy may be sometimes absent, or so slight as to escape observation. The disease begins in early adolescence, and there is a strong tendency to heredity. The muscles first affected are those of the shoulder-girdle. As a rule, degenerative electrical reactions are absent, but this is not invariable. Fibrillar tremors are conspicuous by their absence. Erb's description is based on the clinical observation of twenty cases. Lichtheim,[2] however, has recorded a case rather closely resembling Erb's, in which absolutely no disease of cord or nerves were found.

Schultze's[3] cases are very interesting. There is a marked heredity. Maternal grandfather and grandmother were cousins, but quite healthy; of their children, one son had muscular atrophy of some sort; a daughter, who was quite free from any nervous or muscular affection herself, and was married to a man equally healthy, had five sons, three of whom have muscular atrophy; one died, and an imperfect record is given of the other two. In both the disease began at about eight years. The elder, in whom the disease was less advanced than in the younger, presented the "clinical picture" of pseudo-hypertrophic paralysis, that is to say, the disease began in the calves, which were much hypertrophied. He had the characteristic gait, and difficulty in rising from the ground. But there was also atrophy of other muscles, particularly about the shoulders. The younger brother showed a much more wide-spread atrophy, but without any hypertrophy, and presented the appearance described by Erb.

[1] Erb, "Ueber die Juvenile Form der progressive Muskelatrophie."—'Deutsches Arch. für Klin. Med.' Bd. xxxiv., 1884.

[2] Lichtheim, "Progress. Muskelatroph. ohne Erkrankung der Vorderhörner des Rückenmarks."—'Arch. für Psychiatr.,' 1878, p. 521. Abstract in 'BRAIN,' Vol. II. p. 142.

[3] Schultze, "Hereditäre Muskelatrophie und Pseudohypertrophie der Muskeln." —'Neurolog. Centralblatt,' 1884, p. 529.

Zimmerlin[1] records two families affected with myo-atrophy, much resembling Erb's form, but in one case there was reaction of degeneration in several of the atrophied muscles, and in another there had been fibrillar tremors in the earliest stage of the disease.

Marina[2] gives two cases of what he considers the juvenile form, but without heredity or hypertrophy. In one of his cases there is a partial reaction of degeneration (KCC = ACC) in the deltoid. Marie and Guinon[3] endeavour to show the close connection between the various forms of myopathy. Their first case is one which resembles pseudo-hypertrophic paralysis, but has no marked atrophy of muscles, nor any hypertrophy; but there was progressive weakness of leg-muscles, with the characteristic gait. At the same time the face-muscles seem to have been affected. The second case is one of Erb's form, and the last two belong to the "infantile form," to be described later.

Erb[4] gives an account of a case which he claims to be of the "juvenile form," but in which the muscular affection made its appearance after a fall on the back.[5] He excised a piece of muscle from the deltoid, which was hypertrophied, and another piece from the biceps, which was much wasted. In the former he found the muscle-fibres to be nearly all increased in size, very few showing signs of atrophy. The nuclei were much increased in number round the fibres, and there was also an increase of connective tissue. The vessels were thickened and had more nuclei in their walls than normal. The broadened muscle fibres showed no signs of fatty or granular degeneration, but their striæ were rather faint. In the atrophied biceps what fibres there were left appeared to be hypertrophied, and there was the same increase of nuclei as in the deltoid.

A most important study of muscular wasting is that of Schultze,[6] to which we have already referred several times. The work is divided into two parts, the special and general. The special part relates to a case which was one reported by Friedreich in his monograph on Progressive Muscular Atrophy. The history extends over a period of twenty years. There was a general widespread atrophy of the muscles of the upper extremities, including those of the hands, but also hypertrophy of some of the leg-muscles. There was affection of the diaphragm, but no bulbar symptoms. When

[1] Zimmerlin, "Ueber hereditäre (familiäre) progress. Muskelatroph."—'Zeitschr. für Klin. Med.,' 1884, p. 15. Abstract in 'Brain,' Vol. VII. p 285.

[2] Marina, "Uno studio sulle amiotrophie."—'Lo Sperimentale,' 1885.

[3] Marie et Guinon, "Contrib. à l'etude de quelques-unes des formes cliniques de la Myopathie progressive primitive."—'Rev. de Méd.' 1885, p. 794.

[4] Erb, "Muskelbefund bei der Juvenilen Form der Dystrophia Muscularis progressiva."—'Neurol. Centralbl.,' 1886, p 287.

[5] It is difficult to understand the connection between traumatism and the consequent occurrence of myopathic atrophy and hypertrophy. That such a connection does exist is shown by Eulenburg's case, recorded in the 'Deutsche Med. Wochenschr.,' 1885, p. 177, in which marked hypertrophy of the lower extremities, among other symptoms, followed a fall on the back.

[6] Schultze, 'Ueber den mit Hypertrophie verbundenen Progressiven Muskelschwund und ähnliche Krankheitsformen.' Wiesbaden, 1886. Abstract in 'Neurol. Centralbl.,' 1887, p. 13.

seen by Friedreich, fibrillar tremors were noted in the upper limbs, but none have been seen since. Reaction of degeneration was found in some of the atrophied muscles, such as the left deltoid, thenar muscles, and some interossei. The physical signs here are more in favour of the diagnosis of a myelopathy than that of a myopathy, and yet the anatomical appearances show that the case belongs to the latter class. Schultze found the nervous system, central and peripheral, quite normal, even the small intra-muscular nerves. The small blood-vessels in the nerves showed thickening of the intima, and in the brachial plexus and median nerve they were quite obliterated. The author does not claim anything peculiar to the disease in the occluded vessels, which he regards as due to general endo-arteritis. In the muscles important changes were found. He begins his description with the microscopical appearance of a muscle which appeared quite healthy to the naked eye. The muscle fibres in transverse section appeared angular, some were hypertrophied, but most of them were natural in size. There was an increase of sarcolemma and muscle nuclei, but, as a rule, very little increase of interstitial connective tissue. There was some fatty infiltration in the interstices between the fibres. Here and there were what looked like giant cells filled with smaller cells. These were the remains of muscle fibres, in which the true muscle substance was invaded by the proliferated muscle and sarcolemma nuclei. In muscles showing the highest degree of atrophy, and in which the reaction of degeneration had been made out, large infiltration of fatty tissue was seen, with a number of broad connective tissue fibres, in the meshes of which lay groups of nuclei, indicating the position of destroyed nuclei fibres. In a longitudinal section these nuclei appeared as long rows parallel to one another, sometimes consisting of a chain of forty nuclei.

In some of the muscles, especially the deltoid, the muscle fibres were undergoing a process of vacuolisation. Some fibres presented a sieve-like appearance from the numbers of small vacuoles, others had one large vacuole in the centre.

Dreschfeld[1] gives the results of an autopsy on a case which he believes to be Erb's myopathy. The brain and spinal cord were perfectly normal, as also were the intra-muscular and large-nerves. The muscles showed simple atrophy of the fibres, with some increase of muscle nuclei.

Otto Buss[2] relates the case of two children, brother and sister, æt. 16 and 13 years, respectively. Father and mother are cousins. The author remarks that the boy has a stupid expression, but does not refer it to any affection of the facial muscles. Weakness was noticed first in the legs, æt. 10, but the shoulder girdle-muscles were soon found to waste. Hypertrophy was noted in part of the deltoid and triceps. No reaction of degeneration, or fibrillar

[1] Dreschfeld, "On some of the rarer forms of Muscular Atrophies."—'Brain,' July 1886.

[2] Otto Buss, "Zur Lehre von der Dystrophia muscularis progressiva."—'Berlin Klin. Wochenschr.' 1887, No. 4.

tremors. The girl showed a similar condition, but in a less degree. Some muscle was excised from the boy's biceps, and widening of the muscle fibres, together with increased sarcolemma nuclei, was noted. There was also increase of interstitial connective tissue.

Edgren[1] describes two cases, males, in whom the disease appeared at the ages of 16 and 22. They seem to belong to this type, but there was no heredity in either case.

This appears to be the best place to mention the conclusion of Roth,[2] based on autopsies of myopathies. He says that in excised portions of atrophied muscles he has long noticed that the individual fibres seem to end in long connective tissue fibres. This suggested to him the idea, that muscle fibres might atrophy in their length, perhaps, more than in their breadth. This, he maintains, is borne out by the clinical appearance of the atrophied muscles, the belly of the muscle becoming shorter and shorter. In one muscle that he examined, the zygomatic, he found that half the muscle consisted of tendinous fibrous material, the other half being natural. In a long muscle like the biceps, after suitable hardening processes a fibre could be stripped from one end to the other of the muscle, and then it was found that the part of it remaining of proper muscle tissue was only about 10–20 mm. long, while the rest was of pure fibrous tissue. He believes this to be peculiar to atrophies of myopathic origin.

Singer[3] gives an account of two complete cases, one of which belongs to this form, and the other to that of the type to be next described, showing how closely the two forms are allied. The first is that of a woman aged 34, single, with no heredity of a neurosis. The first symptoms were weakness of the lower extremities, with pseudohypertrophy of calves, and characteristic gait. This was followed by wasting of muscles generally. The shoulder girdle muscles were much atrophied, so also the upper arm muscles, but not the small hand muscles. No fibrillar tremors or reaction of degeneration.

Post-mortem.—The atrophied muscles showed general lipomatosis. There was some diminution in size of a small part of the lumbar grey matter on the left side, together with corresponding decrease in the number of ganglion cells, especially in the lateral horn, where on the right side there were twenty-six, and on the left three. Other nerves quite normal. The autological appearances of the muscles consist of increase in the interstitial connective tissue, splitting up of the fibres in a longitudinal direction, with also a tendency to split into plates horizontally, as occurs in every degeneration. The nerves were absolutely normal.

In the second case, the face muscles were affected on the left side, otherwise the case resembles the last, particularly in the

[1] Edgren, "Om den primära progressiva myopatin, etc."—'Nord. Med. Arkiv,' Bd. xix. Nr. 5.

[2] Roth, "Contribution à l'anatomie pathologique de l'atrophie musculaire progressive."—'Comptes Rendus de la Soc. de Biologie,' Dec. 17, 1886.

[3] Singer, "Zur Kenntniss der primären Myopathieen."—'Zeitschr. für Heilkunde,' Bd. viii. p. 229.

matter of the histological appearance of a piece of muscle excised for the purpose. Both these cases obviously are closely allied to pseudo-hypertrophic paralysis.

We now come to consider another distinct type of myopathy. Duchenne was well acquainted with a peculiar form of progressive muscular atrophy, in which the face muscles were early affected, and which so constantly appeared in childhood, that he called it the "Infantile form" of progressive muscular atrophy. The mode of attack is so characteristic, that instances of the disease may be traced in many of the descriptions of the earlier writers.

Landouzy and Déjérine[1] contribute a very valuable addition to our knowledge of this form. The first-named observer was so fortunate as to have been able to watch two brothers for a period of ten years.[2] In another family affected with the same disease, the authors were able to make a post-mortem examination on one of the members, by which it was established that the disease is purely myopathic, the nervous system, central and peripheral, being absolutely normal. In this disease there rarely if ever occurs any hypertrophy of muscles, atrophy is the predominant feature. The group of muscles first affected are those of expression; then soon follows atrophy of the shoulders and upper arms, the small hand-muscles being affected very late in the course of the disease. The muscles of the lower extremities may be affected sooner or later. As in the type last considered, fibrillar tremors are almost never present, and degenerative electrical reactions are rare. As the facial affection is peculiar, it will be well to describe it here. The forehead becomes smooth and quite devoid of wrinkles, even during vigorous alterations of expression, such as laughing and crying. Owing to wasting of the orbiculares palpebrarum the eyes are never quite closed, even during sleep. The lips are everted from wasting of the orbicularis oris, and the levatores labii superioris, but the zygomatics and risorius seem to escape, so that in laughing the mouth extends laterally (*rire en travers*), the upper half of the face not sharing in the change of expression. The muscles of mastication and deglutition escape entirely. The whole appearance is termed the "facies myopathica" by the authors.

In Remak's[3] case, which appeared at nearly the same time as Landouzy's preliminary communication, the face muscles were affected after the shoulders and upper-arm muscles. He considers it to be one of the juvenile type of Erb.

Mossdoff[4] records a similar case to Remak's, but with asymmetry

[1] Landouzy et Déjérine, "Myopathie atrophique progressive, sans Neuropathie."—'Rev. de Méd.,' Feb. 1885. A preliminary paper by Landouzy appeared in 'Comptes Rendus de la Soc. de Biol.,' 1884.

[2] Landouzy, "Deux cas de l'atrophie musculaire progressive de l'enfance."—'Comptes Rendus de la Soc. de Biol.,' 1874.

[3] Remak, "Ueber die gelegentliche Betheiligung der Gesichts-Musculatur bei der Juvenilen Form der progr. Muskelatrophie."—'Neurol. Centralbl.,' 1884, p. 337.

[4] Mossdorf, "Ein zweiter Fall von Betheiligung der Gesichts-Musculatur bei der Juvenilen Muskelatrophie."—'Neurol. Centralbl.,' 1885, p. 1.

of the facial affection. Both these cases correspond to the descriptions of Landouzy and Déjérine.

Charcot's[1] case illustrates well the facial affection in this disease. He insists strongly on the close analogy between the three forms of myopathy, pseudo-hypertrophic paralysis, Erb's form, and the form now under consideration.

Westphal's[2] third case is one of facial myopathy, in which there is a marked atrophic heredity, younger sister, father, and uncle.

Cenas and Douillet[3] record three cases in the same family, two brothers and sister; the eldest appears to be a case of the type now under consideration. The next was the sister, in whom the atrophy commenced in the thenar muscles of the right hand, and that after suffering for six months or more from severe neuralgic pains in the arm. She had previously, some six years before being seen, had suppurative synovitis of the right index finger. This may have been the determining cause of the atrophy, but however that may be, the case reads like one of the Aran-Duchenne type. The last case, another brother, presented the facial appearances of a myopathy, but fibrillar tremors were seen in some of the muscles. The existence in the same family of myopathy and myelopathy is very interesting, and would point to a possible common origin for both. In this connection may be mentioned Philip's[4] observations. Here we have a family of eight children, four of whom were affected with pseudo-hypertrophic paralysis of the most marked description. On the mother's side there were a cousin and an uncle "paralysed." The father's family history was good, but he himself suffered from a form of spastic paralysis, with wasting and contracture of muscles, which seems to be undoubtedly of spinal origin, but which had probably connection with an accident.

In Ladame's[5] case, the grandfather on the father's side was said to have died of muscular atrophy. The patient was a young man in whom the disease commenced at the age of twelve, after typhoid. The muscles first affected were those of the shoulder and upper extremities, the face muscles being attacked later. Leg muscles were much wasted, knee-jerk abolished. There was no hypertrophy, or reaction of degeneration.

Westphal[6] records three cases, mother, daughter, and mother's

[1] Charcot, "Révision Nosographique des Atrophies Musculaires progressives." —'Progrès Médical,' 1885.

[2] Westphal, 'Berlin Klin. Wochenschr.,' 1885, p. 617.

[3] Cenas et Douillet, "Deux cas de myopathie atrophique, type Landouzy-Déjérine, et un cas d'atrophie musculaire, type Aran-Duchenne, dans la même famille."—'Loire medicale,' 1885, Nos. 7, 8.

[4] Philip, "Primary Spastic Paralysis, and Pseudo-hypertrophic Paralysis in different members of the same Family, with probable Heredity in both."—'Brain,' Vol. VIII. p. 520.

[5] Ladame, "Contribution à l'étude de la myopathie atrophique progressive."—'Rev. de Méd.,' 1886, p. 817.

[6] Westphal, "Ueber einige Falle von progressive Muskelatrophie mit Betheiligung der Gesichtsmuskeln."—'Charité Annalen,' xi., 1886. Abstract in 'Neurol. Centralbl.,' Jan. 1887.

sister, evidently of this type of myopathy. The face muscles were markedly affected in all.

Landouzy and Déjérine[1] add six more cases to the number.

Obs. I. is a very characteristic case of the disease. The facial peculiarities with atrophy of the shoulder muscles existed in the mother, the maternal grandmother, also in the younger brother.

Obs. II., a young woman, æt. 27, had the "facies myopathica" well-marked, but there was no heredity, no fibrillar tremors, or hypertrophy; but there was reaction of degeneration in the orbicularis oris, and the flexors of the left forearm. The interest in Obs. III. lies in the fact that his brother was seen, described, and photographed by Duchenne in 1872, as an instance of the infantile form of progressive muscular atrophy. In Obs. IV. the face muscles were affected late in the disease, and degenerative reactions were found in several muscles. Obs. VI. was one of the scapulo-humeral type, but without the facial characteristics. The author made a post-mortem examination on this case, and found that the nervous system, central and peripheral, was perfectly normal. In the muscles they found appearances closely resembling those described by Schlutze (op. cit.). They consider these appearances to be due to an "irritative myositis."

Kreske[2] records a typical case of this disease, the facial symptoms dating from three years old. No hypertrophy, or reaction of degeneration.

From the foregoing cases and opinions of writers, it is easy to see that a very close analogy exists between the three myopathies, pseudo-hypertrophic paralysis, the "Juvenile form," and the "Infantile form." The first and third are clinically very unlike each other, but the second stands as a transition form between the two. For instance, hypertrophy is the predominant feature in the first, a very subordinate one in the second, and absent in the third. The second and third may so resemble one another as to be undistinguishable but for the facial symptoms in the latter. All three are diseases of youth, and all are very strongly open to hereditary influence. As far as they have been yet studied, the same essential morbid processes are at the seat of each. And yet with all these points of resemblance, they are still distinct nosological types. It is to be hoped that in the near future they may receive some less clumsy and misleading names than they do at present; the name "facio-scapulo humeral type of myopathy," suggested for the last by the French authors, scarcely recommends itself either on the score of elegance or brevity.

Among the hereditary forms of progressive muscular atrophy, it has long been noticed that the disease may commence in the

[1] Landouzy et Déjérine, "Nouvelles recherches cliniques et anatomo-pathologiques sur la myopathie atrophique progressive."—'Rev. de. Méd.,' 1886, p. 977. Abstract in present No. of 'BRAIN.'

[2] Kreske, "Ueber die myopathische Form der progressive Muskelatrophie Betheiligung der Gesichtsmuskeln."—'Munchener Med. Wochenschr.,' No. 15, 1886. Abstract in 'Neurol. Centralbl.' 1886, p. 302.

lower extremities. This has been observed by Bamberger,[1] Friedreich and Leyden. It must be remembered that pseudo-hypertrophic paralysis generally commences in that way, but the form now to be considered has no relation to that disease at all. This form was recognised as a distinct type by Charcot and Marie[2] in February 1886, and almost at the same time, and quite independently, by the writer of this review.[3] The latter proposed for it the provisional name of the "peroneal type of progressive muscular atrophy," because though it may attack any group of the lower leg muscles first, yet it seems to commence oftener in the peroneal group than in any other.

In this form heredity is a marked feature; the disease generally makes its appearance in childhood. Though the leg muscles are first affected, Charcot and Marie notice that the small hand-muscles are nearly certain to be affected sooner or later. A very common early symptom is the appearance of talipes varus, due simply to loss of power in the peronei, not to contraction of opposing muscles; this talipes is very gradual and progressive in its development, unlike that due to infantile paralysis. The affected limbs are cold, and often tend to become livid from disturbance of vaso-motor influence. Cramps and pains in the limbs are of frequent occurrence. Fibrillar tremors may or may not be present. Reaction of degeneration, as a rule, makes its appearance sooner or later. In the thesis above mentioned, the writer has collected some twenty recorded cases from various sources, which appear to be illustrations of this type. Among these are accounts of four post-mortems by Virchow,[4] Oppenheimer,[5] and Friedreich,[6] which show that disease of the cord is absent in two, and in the other two what disease there is, is probably secondary, being situated in the posterior columns. On the other hand, in three of these autopsies was found a well-marked interstitial neuritis affecting the nerves supplying the atrophied muscles. It is therefore probable that this type is a true *neuropathy*, using the term neuropathy in its restricted sense.

Among the later recorded cases which appear to belong to this form are those of Osler, Schultze, Ormerod, and Charcot and Marie.

In Osler's[7] case heredity is remarkably shown, and can be traced backwards through three generations. The disease began unusually late in life, æt. 47, but the symptoms seem fairly

[1] Bamberger, "Bemerkungen über progr. Muskelatroph."—'Oesterr. Ztg. für prakt. Heilk.,' No. 7, 1860. Abstracts in Canstatt's Jahresb., 1860, p. 88.

[2] Charcot et Marie, "Sur une forme particulière d'atrophie musculaire progressive," etc.—'Rev. de Méd.,' 1886, p. 97

[3] Tooth, "The peroneal type of progressive muscular atrophy." Graduation Thesis, M.D. Cambridge, 1886.

[4] Virchow, "Ein Fall von progr. Muskelatrophie."—'Virch. Arch.,' 1855.

[5] Oppenheimer, "Ueber progr. fettige Muskelentartung." Heidelberg, 1855. Abstract in Canstatt's Jahresb., 1855.

[6] Friedreich, "Ueber progr. Muskelatroph.," 1873. Cases I. and II.

[7] "On Heredity in Progress. Musc. Atroph., as illustrated in the Farr family of Vermont."—'Arch. of Med., N.Y.,' 1880.

characteristic. Schultze's[1] cases are those of two sisters and a brother, æt. 3, 5, and 9 years respectively. In them the disease commenced in the peronei, at 8 years, leading to very early and gradual appearance of talipes varus. In the eldest the thenar muscles were affected early.

There were no fibrillar tremors, no pains along nerves or in muscles, but complete reaction of degeneration in many of the muscles. Schultze himself considers the disease to be essentially due to a peripheral lesion. That it is not an ordinary neuritis would seem to be suggested by the collateral heredity.

Of Ormerod's[2] cases, two, a brother and sister, were affected with atrophy, and contraction of the peroneal and calf muscles, soon after measles; the hand muscles were attacked about four years later. There was reaction of degeneration in some of the muscles, but no fibrillar tremors. A very interesting fact concerning them is that the father has had atrophy of the right peroneal and calf muscles since three years old. He has been told that he had measles badly when he was a year old. The atrophic condition has not progressed in his case.

Charcot and Marie (op. cit.) contribute five cases, two of which are brothers. In all, the disease commenced in the leg muscles; peronei or calf, leading three cases to talipes. At a time varying from two to five years after, the hand-muscles were attacked. There was reaction of degeneration of some of the muscles in three of the cases, and fibrillar tremors in all. Cramps of the muscles were very common.

This form of muscular atrophy requires much more attention than it has yet received, and post-mortem examinations are particularly needed.

Seppili[3] tabulates amyotrophies as follows:

A. *Neuropathic.*—1. Peripheral (toxic, infectious). 2. Central (myelopathies). *a.* Progressive muscular atrophy. *b.* Amyotrophic lateral sclerosis. *c.* Deuteropathic spinal amyotrophies (from extension of disease into anterior horns).

B. *Myopathic.*—Progressive muscular dystrophy, or progressive primary myopathy. *a.* Infantile progressive muscular atrophy. *b.* Erb's juvenile form of muscular atrophy. *c.* Muscular pseudohypertrophy. *d.* Leyden's hereditary muscular atrophy. *e.* Transitional forms.

[1] Schultze, "Ueber eine eigenthümliche progress. atroph. Paral. bei mehren Kindern derselben Familie."—'Berlin. Klin. Wochenschr.,' No. 41. 1884.

[2] Ormerod, "Muscular Atrophy after Measles in three members of a Family." —'BRAIN,' Vol. VII.

[3] Riv. Sper. di Fren., p. 120, 1887.

BRAIN AND EYE—OPTIC NEURITIS.

BY JAMES ANDERSON, M.D.

It is now six years since the classical discussion on Optic Neuritis in Intracranial Disease took place during the first session of the Ophthalmological Society. Much work, clinical, microscopic and experimental, has since then been done in elucidation of the subject; but it cannot be regarded as much less of a *quæstio vexata* now than it was six years ago. Another more purely ophthalmological question has also been under discussion during these years, viz. Sympathetic Ophthalmitis. Notwithstanding important differences, the two questions have much in common, and it seems probable that when the one has been solved, the other will present but little difficulty. While in both cases the ultimate decision must rest on clinical observation, in both cases experimental observation can do much in clearing the ground, and in suggesting at least a *vera causa*. To Deutschmann, of Göttingen, we are indebted for experimental work on both subjects, and we purpose giving some account of his recent monograph on "Optic Neuritis in connection with Brain Disease,"[1] with some reference to the work of others, confirmatory and opposing.

Observers are now, and indeed they were six years ago, practically unanimous in regarding optic neuritis as a single process, not distinguishable into "choked disc" and "descending neuritis," as was formerly made out. It is clear on reading various descriptions of what were considered the distinguishing features of "choked disc," that there was no general agreement on the point. The term implied a theory, but expressed no clinical fact, and it has simplified matters that it is now practically obsolete. At the same time it would be very far from right to conclude that there are not types of neuritis. The neuritis of tubercular meningitis, for example, is as a rule of quite a different type from that of cerebellar tumour. The low, pinkish-white swelling of the first is very

[1] Jena, Gustav Fischer, 1887, p. 68. For full Abstract v. 'Ophthal. Review,' April, 1887.

distinct from the high, blood-coloured and generally blood-stained swelling of the other. Both however, are almost certainly but different intensities of a similar process, and probably the main element in the difference is the "choking," which, as shown by Gowers in the above-mentioned discussion, does actually take place, not at, but in front of, the lamina cribrosa.

In his paper, opening the discussion on Optic Neuritis, Dr. Hughlings-Jackson referred to three hypotheses regarding the cause of the inflammation: Von Graefe's, Schmidt's, and the reflex vaso-motor theory of Benedict, Brown-Séquard and others. The last hypothesis he considered, and the writer believes he still considers, the most plausible explanation, although he in no way commits himself to it. There are many difficulties in the way of accepting it. Reflex vaso-motor irritation, will, we presume, be readily admitted by all pathologists; but very few, we imagine, would admit that this irritation can proceed to actual inflammation, exudation of lymph, and hæmorrhage, such as occurs in tumour neuritis. The only instance at all parallel with it is that of Sympathetic Ophthalmitis, and the work of Leber, Deutschmann and others has made it highly probable that there is in every such case an infective process by way of the chiasma. The fact mentioned by Dr. Jackson, that tumours may induce discharges in neighbouring grey matter, goes only to show that tumours may cause an instability of grey matter, either by immediate pressure, or by interference with vascular supply, whether directly or reflexly. Von Graefe's theory attributes the neuritis to compression of the cavernous sinus by the tumour, the consequences of this compression appearing specially at the lamina cribrosa from its rigidity. The theory is negatived by the fact, that the ophthalmic and the angular veins communicate so freely that even obliteration of the cavernous sinus will cause no venous stasis in the eyeball or orbit.

Schmidt's theory attributes the neuritis to the pressure of fluid effused in the subdural and subarachnoid spaces, and forced into the vaginal spaces of the optic nerve; and Schultén has recently shown, that the injection of fluids into the subdural and subarachnoid spaces will actually cause narrowing of the retinal arteries and distension of the retinal veins, the changes however, being temporary. Deutschmann has subjected this theory to further experimentation in order to settle the question, what degree of hydrops of the optic nerve in animals is required to produce changes in the disc similar to those of the choked disc in man. With antiseptic precautions he laid bare the optic nerve, divided it, injected the distal part of it with agar-agar solution, and ligatured it,

taking care not to injure the central vessels. With moderate distension, greater however than that produced by cerebral tumour, there was only the temporary vascular change observed by Schultén, while in order to produce swelling and œdema of the papilla, the nerve sheath required to be so forcibly distended as to arrest the circulation in the retina, and even then the nerve showed no true neuritis. Such distension and arrest of circulation is never present in the human subject. Again, repeated injections of agar-agar solution into the cranial cavity of an animal, although they caused considerable intracranial pressure and distension of the optic sheath, in no instance produced inflammation of the papilla, the nerve or its sheaths. These experiments seem certainly to decide against Schmidt's theory.

In 1881 Leber put forward the view expressed in the Transactions of the Medical Congress of that year:[1] "The optic nerve is the path of communication between the affection of the brain and that of the eye. An essential part in this transmission is taken by the effusion of a serous fluid into the sheath of the nerve extruded from the cranium by the increased intracranial pressure. This fluid does not act by simple mechanical pressure, since its quantity is sometimes small; probably it possesses phlogogenic properties." To test this theory, Deutschmann repeated his injection experiments, using an infecting material in place of the agar-agar solution. A solution of common salt containing traces of staphylococcus was injected, in the manner above described, within the optic sheath. During the first day the papilla became red and swollen, the veins distended, the arteries perhaps diminished in size. Next day the papilla was greatly swollen, and the retina turbid; and the microscope showed well-marked neuritis and peri-neuritis. Injections of the same solutions into the skull caused acute inflammatory changes, resulting in death without the eyes being affected, and it was necessary, therefore, to select an infective material of slower action. A few drops of tubercular pus were introduced, with antiseptic precautions, into the subdural space. The animal remained apparently healthy for three weeks, when the papilla became red, and the veins tortuous. True papillitis followed, which either gradually subsided, or went on to post-neuritic atrophy. The microscope showed tubercular meningitis, the intracranial portion of the nerve normal, the orbital portion more or less altered, the sheath being distended more or less with exudation in the early stages, and occupied with tubercle deposits in the later; the nerve and its sheath being both inflamed. Deutschmann considers that this proves

[1] 'Abstracts,' p. 474

Leber's theory as to the cause of neuritis, and he suggests that the phlogogenic elements contained in the fluid secreted in the subdural and subarachnoid spaces are micro-organisms already present in the system, but congregated round the tumour as a place of least resistance. Deutschmann considers that a true descending neuritis is rarely demonstrable, and when a continuous neuritis can be traced, he considers that it is generally an ascending, rather than a descending, neuritis; that the bulbar end of the nerve is affected first, and from this point it travels backward. Where only one eye is affected in intracranial tumour, Deutschmann suggests that there is in the one nerve-sheath some unusual obstruction, either recent, or of older date, preventing the passage of the infective material.

It will at once be seen that the evidence afforded by the injections of tubercular matter into the subdural space is not absolutely cogent, and Edmunds and Lawford point out that,[1] in order to substantiate his point, Deutschmann would require to show that if the animals be killed in the first few hours or days after inoculation, no optic neuritis is present; that at a later stage inflammation confined to the bulbar end of the nerve is found, and that later still the whole length of the nerve is involved. Edmunds and Lawford [1] have recorded the results of their examination of the optic nerves in cases of intracranial disease, and an analysis of cases of intracranial tumour with respect to the existence of optic neuritis. Having examined some forty cases, they found that in every case in which papillitis was seen during life there were inflammatory changes in the optic nerves in their entirety. In a few instances the inflammation was confined to the proximal part of the nerve, and in these the papilla, both to the ophthalmoscope and microscope, showed no changes, death having occurred before the inflammation had time to travel down the nerves as far as the papillæ. They conclude therefore that the optic neuritis which occurs in intracranial disease is due to the presence of a secondary meningitis, and they find themselves strengthened in this opinion by the result of their analysis of cases. Of ninety-six cases of fatal cerebral tumour, optic neuritis was present in eighty-six per cent. of the cases in which the new growth was situated in the basal ganglia or in the cerebellum, whereas in only forty-six per cent. of those cases where the tumour was at the convexity of the brain did neuritis occur. In the discussion on optic neuritis, Gowers stated that it was probable that mere distension of the sheath

[1] 'Ophth. Rev.' May, 1887.
[1] 'Ophth. Soc. Trans.' vol. iii. p. 138, and vol. v. p. 172

might cause papillitis, but he showed by specimens that descending inflammation was almost invariably present, even where there was no naked eye evidence of it. One of his specimens is interesting in relation to Leber's theory. It showed, as usual, inflammation along the length of the optic nerve, but much more intense at the chiasma and behind the globe. From it he argued that a very slight degree of descending inflammation may be the connection between a neuritis of the cerebral, and one of the ocular extremity of the nerve. The optic papilla, as he remarked, is a structure in which inflammation readily occurs, whether from intracranial or from general disease. And it is to be remembered that the papilla may be repeatedly and severely inflamed. The writer has recorded a case of relapsing neuritis[1] where a patient recovered with perfect vision from two separate and severe attacks of neuritis. Since the case was recorded the patient has had a third attack similar to the others, and has again recovered with perfect vision. Such a case is somewhat difficult of explanation on Leber's theory alone; but Leber's theory does not actually contradict the theory of a descending neuritis, or peri-neuritis, as advocated by Edmunds and Lawford. It is possible, perhaps it is probable, that both views are correct, each cause playing a part, more or less, in different cases.

[1] 'Ophth. Rev.' May, 1886.

Reviews and Notices of Books.

The Functions of the Brain. By DAVID FERRIER, M.D., LL.D., F.R.S. Second edition. Rewritten and enlarged. London: Smith, Elder, and Co., 1886.

IT has been understood for some time by those who take even a general interest in neurological studies, that Dr. Ferrier has been engaged in preparing a second edition of his celebrated treatise on the 'Functions of the Brain'; and now that the work has made its appearance, it will receive a hearty welcome both from the profession and the educated public. In its external appearance, the new book is an almost exact reproduction of its predecessor, the green-coloured back of which formed such a prominent and well-known feature in the library of every educated physician. The new edition extends to 498 as against 323 pages in the old. If we consider that the author gives, not only a detailed account of the numerous researches he has himself undertaken in the ten years elapsed since the publication of the first edition, but also a systematic exposition of the results obtained by other investigators in the field of neurology during this period, it will be at once seen that he has exercised a marvellous amount of self-restraint in not increasing the size of his book by more than 175 pages; and when we come to examine the contents of the work, this self-restraint becomes strikingly apparent. The first chapter deals with the structure of the cerebro-spinal centres, and there is not a page of the fifty which it comprises in which the instructed reader will not observe the temptation under which the author must have laboured to branch out into discussions about contested points. Such discussions are useful and necessary under certain circumstances, but Dr. Ferrier judged, as we think wisely, that his book was not the proper place for them. He accordingly proceeds directly to his object, and gives a succinct and transparently clear account of the structure of the nervous system—an account which is as accurate and trustworthy as can possibly be imagined within the limits he has imposed upon himself.

Before proceeding further, we may notice the beauty of the original woodcuts which illustrate this chapter. The new edition is illustrated by 137 figures as against 68 in the old. All the illustrations are good; but the representation of sections of a lobule of the cerebellum by Dr. Beevor (Fig. 21), and of the medulla oblongata, pons, and corpora quadrigemina by Dr. Bevan Lewis (Figs. 14, 15, 16 and 19), are models of anatomical drawings, while the representations of sections of the cerebral cortex by Dr. Bevan Lewis (Figs. 28 to 33) are altogether unique,

and probably excel, in delicacy of drawing and beauty of execution, anything we have ever seen produced by the wood-cutter's art for the purposes of histological illustration. It is only those who have had the privilege of examining sections of the cortex cerebri, as prepared by Dr. Lewis or by one of his pupils, who can at all appreciate how wonderfully accurate and faithful these drawings represent the original sections. We cannot help expressing some regret, that these beautiful figures are not accompanied by a brief description of the histology of the cortex, even at the risk of adding a few more pages to the size of the book; but the omission is only another striking example of the self-restraint already spoken of, as being exercised by the author throughout every part of the work.

In the second chapter the author treats of the functions of the spinal cord. An account is given first of the course of the conducting paths of the cord, as determined by embryological and pathological observations as well as by experiments on animals; and here the author favours the opinion that the sensory paths lie, not in the posterior, but in the lateral columns, in the limiting zone of Flechsig. He admits, however, the difficulty this opinion encounters in the fact, that these areas do not undergo an ascending degeneration when the cord is divided transversely, and endeavours to surmount it by supposing that these tracts maintain a continuous connection with the cells of the posterior horns. But in that case the sensory fibres belonging to the lower extremities must be connected with the cells of the posterior horns throughout the whole length of the cord, and it is not clear how they can at the same time be situated in the limiting zone of Flechsig. The author believes that the muscular sense is not a simple sensation, but one formed by a combination of the common forms of cutaneous and muscular sensibility. He asserts that "the sensations arising in connection with muscular action are of a purely centripetal character," and utterly rejects Brown-Séquard's hypothesis, that the paths of the muscular sense pass in the anterior roots and motor tracts of the spinal cord.

The action of the spinal cord as an independent centre is next discussed; the functions of reflex activity, co-ordination of synergic movements, muscular and vascular tonus, trophic regulation, and the regulation of the movements of special organs like the bladder, rectum, sexual organs and iris, being passed in review. The remarkable sobriety of Dr. Ferrier's judgment in interpreting the phenomena resulting from destructive and irritative lesions of various parts of the nervous system, is apparent in his remarks on the experiments of Tscheschichin, Burdon-Sanderson, Wood, Ott, Aronsohn and Sachs, Eulenberg and others on the alteration of the temperature of the body which is caused by injuries of the spinal cord, medulla, pons, and certain parts of the brain. "The facts," he says, "only allow us to conclude that injuries of certain centres and tracts cause increased heat production, but the mode of operation is by no means satisfactorily indicated;" and "it by no means

follows that we are to assume the existence of a special heat-producing centre, distinct from those which influence the motor and vaso-motor apparatus in general."

In the next chapter, which deals with the functions of the medulla oblongata, the author throws out a very important suggestion with the view of explaining the devious course pursued by the vaso-motor nerves, and the dilator nerves of the iris, which, arising from their centres of origin in the medulla, descend through the whole length of the cervical part of the spinal cord to emerge with the upper dorsal nerves, whence they pass into the cervical sympathetic. He believes that the medulla ought to be regarded as the direct continuation of the dorsal spinal cord, and that the cervical portion is intercalated in the course of development for the innervation of the upper extremities. Many well-known embryological facts support this hypothesis, and if its truth be granted, an adequate explanation would be found for facts which are otherwise inexplicable; such as the course of the cardiac excito-motor branches of the sympathetic, the extraordinary path pursued by the spinal portion of the spinal accessory nerve, and the origin of the ascending root of the fifth nerve being as low down as the level of the fifth cervical pair of nerves.

The author next treats of the formations of the mesencephalon and cerebellum in a general manner, and classifies the functional manifestations of these organs under the heads of the maintenance of equilibrium, the co-ordination of locomotion and emotional expression. His discussion of the influence of tactile, visual, and labyrinthine impressions on the maintenance of the equilibrium and of the disturbances of equilibration caused by lesions of those impressions is exceedingly able, and demands a careful study. Dr. Ferrier now attempts to determine the functions of the different centres of which the mesencephalon and cerebellum are composed, but this task is so inherently difficult and complicated that it need not be wondered if it is only attended by partial success.

From the results of both irritative and destructive lesions of the corpora quadrigemina, he concludes that these ganglia "form an essential portion of the mechanism of the co-ordination of retinal and general sensory impressions with the mesencephalic motor apparatus concerned in the responsive adjustments of equilibration and the other adaptive reactions of which animals are capable after removal of the higher encephalic centres."

In considering the functions of the cerebellum, Dr. Ferrier passes in review the classical experiments of Flourens, and gives due weight to the staggering gait and other disorders of equilibration occasioned by destructive lesions of the organ, whether resulting from experimental injury or disease; but the most remarkable light on the functions of the organ is probably to be found in the definite movements of the eyes, head, and limbs which the author obtained from electrical stimulation of the cortex. It is, indeed, hardly possible to over-estimate the theoretical and practical value of Dr. Ferrier's experiments on the cerebellum, and it seems to us strange that they appear never to have been repeated by any

other physiologist, either for the purpose of testing the accuracy of the results already obtained, or of eliciting new facts by extending the scope of the experiments themselves. The author justly observes, that the ocular and other movements resulting from electrical excitation of the cortex of the cerebellum confer great significance on the phenomena observed in man, when a galvanic current is passed transversely through the skull in the cerebellar region. When the positive pole is placed in the right mastoid fossa and the negative in the left, the head and body sink towards the positive pole when the circuit is closed, and external objects seem to whirl to the left, or in other words, there is a conjugate deviation of the eyes to the right. The author seems, however, to have overlooked the similarity of these phenomena to those produced by pressure on the tragus of the ear, in two cases of Menières' disease reported by Dr. Hughlings-Jackson. In a case of the kind under the writer's observation at present, the patient, a young man aged 17 years, on putting the tip of the index finger of his right hand into the external meatus of his right ear, staggers to the left and backwards; there is at the same time a strong conjugate deviation of his eyes to the left, followed by a rapid oscillation of the eyeballs, the movements being accompanied by a subjective feeling of objects whirling to the right. The similarity of these phenomena to those resulting from passing a galvanic current through the head is too obvious to require pointing out. It seems probable therefore that in the galvanic experiment the current acts, not directly on the cortex of the cerebellum itself, but on the semicircular canals, and it is also probable that the negative pole is, as usual, the stimulating one, inasmuch as in our patient the inclination of the head and the drawing of the eyes were away from the ear which was the subject of irritation. But whatever view may be taken of the manner in which transverse galvanisation of the skull produces these ocular movements, it does not in the slightest degree detract from the great value of the results obtained by Dr. Ferrier from electrical excitation of the cerebellar cortex.

The author now comes to the consideration of the functions of the cerebrum, which forms by far the most important part of the work. Nevertheless, we do not propose to discuss the author's views on this subject at any great length. Our reasons are that his views, which were the subject of warm controversy ten years ago, are now almost universally accepted. Differences of opinion there may be with regard to minor details, but the great principles of cortical localisation are adopted by all neurologists, and daily acted on practically at the bedside. After describing the methods of investigation, Dr. Ferrier gives a detailed account of the phenomena resulting from electrical excitation of the cerebral hemispheres. The most important addition in the new edition as compared with the old, in this part of his subject, is to be found in the description of the movements observed by Horsley and Schafer on excitation of the marginal convolution. These movements are, speaking broadly, caused by contractions of the muscles of the trunk, such as the thoracic, lumbar, and abdominal muscles, and of

those muscles of the limbs and head which have their origin in the bones of the trunk, such as the muscles which produce movements at the hip and shoulder joints and of the head. The movements in question are those which we have in common with the largest number of animals, and they are consequently the most general movements of the body, while the movements which are peculiar to man, such as those of articulation and of the hand in writing and in playing the piano, are organised in or near the operculum. Such a distribution of the localisation of the general and special movements of the body is not accidental, and an ample explanation of it will doubtless be found in the comparative anatomy and embryology of the brain, but into this wide subject we cannot at present enter. Other noteworthy facts which come out in this part of the enquiry are, that the fibres of the conducting paths which connect the motor area of the cortex and the spinal nuclei are excitable, and functionally differentiated like the cortical centres, whether they be examined in their passage through the centrum semi-ovale or in the internal capsule. The author has also found, that excitation of the corpus striatum causes tonic spasm of the opposite side of the body; while excitation of the optic thalamus is not followed by any motor manifestation.

Coming now to the interpretation of the motor reactions obtained on electrical stimulation, Dr. Ferrier believes that some of these movements indicate that the part of the grey matter irritated is directly connected with the motor strands of the crus cerebri and spinal cord, while others of the movements only indicate that the part stimulated is indirectly connected with these strands, the direct effect of the stimulation being some modification of consciousness. In accordance with this view, the author divides the excitable portion of the cortex into a motor and a sensory area, but which part is to be regarded as belonging to the one and which to the other, can only be decided by destructive lesions of the respective centres. If the centre is a motor one, its destruction will give rise to a paralysis of a definite group of muscles, and if sensory to some defect of common or special sensation.

Whatever theoretical objections may be urged against this division of the functions of the excitable area of the cortex, there can be no doubt that, regarded as a practical distinction, it has proved of immense value in Dr. Ferrier's hands, and one can only marvel how wonderfully near the truth his first surmises with regard to the localisation of the sensory centres have turned out to be. The visual centre, the localisation of which has been a subject of keen controversy, is now stated by Dr. Ferrier to be situated in the occipito-angular region, and not in the angular gyrus, as he first supposed. The author found that destruction of one angular gyrus caused temporary amblyopia of the opposite eye, while destruction of both gyri caused complete blindness, which partially disappeared in a few days. Vision was not appreciably affected by destruction of one or even of both occipital lobes. Destruction of the angular gyrus and occipital lobe in one hemisphere caused transient amblyopia in the opposite eye, and a more

or less enduring hemiopia in both eyes towards the side opposite the lesion, from paralysis of the retinæ on the same side as the lesion. Destruction of the occipito-angular regions in both hemispheres caused complete and permanent blindness. From these experiments, which are a model of cautious inductive enquiry, the author concludes that each angular gyrus is associated with both eyes, but chiefly with the opposite one, and that, through the occipito-angular region, each hemisphere is associated with the corresponding halves of both retinæ, thus proving that the semidecussation of the optic fibres which takes place in the chiasma is maintained in the cortical centres. Such being the author's conclusions, it is manifest that he is compelled to reject Charcot's scheme of the decussation of the optic tracts, as indeed we believe the distinguished author of that scheme has himself already done, with that freedom from bias and ready appreciation of new facts which characterises him. The scheme which Dr. Ferrier proposes to substitute for that of Charcot, and the less known ones of Grasset and of Sharkey, has the demerit of being somewhat complicated, but it is those who are unaccustomed to investigate nature that are most ready to talk about "the simplicity of nature's ways." Nature's ways, especially in cerebral physiology, are exceedingly complicated, and it is possible that there is no way of graphically representing the facts by a simple diagram; and the one proposed by Dr. Ferrier possesses the merit of representing the facts ascertained, both from experiments on animals and from pathological observation, with tolerable accuracy, and far better than any scheme which has hitherto been constructed. At the same time, we do not believe, and Dr. Ferrier would be the last to maintain, that we have yet had the final word on the localisation of the visual centre. It is probable that the recent observations of Spencer on the pineal eye of the Lacertilia is destined to throw a new light upon the structure of the visual central nervous mechanism, and each addition to the accuracy of our knowledge of the structure of nerve mechanisms will render more definite our knowledge of their functions.

In his previous edition Dr. Ferrier placed the centre for tactile and common sensation in the hippocampal region, but the recent observations of Horsley and Schafer have proved that the gyrus fornicatus forms part of this centre. It has already been pointed out by Broca that the gyrus fornicatus and the gyrus hippocampus form one lobe, which he named the *falciform lobe*, and which he erroneously believed to be related to the sense of smell, but it now appears that this lobe forms the centre for tactile and common sensation. Whether it is or is not likewise the centre for the muscular sense remains to be determined. The localisation of the auditory centre in the superior temporo-sphenoidal convolution, proved by the early experiments of the author, and afterwards confirmed by pathological observations, is now universally acknowledged. Nothing is known with regard to the olfactory and gustatory centres beyond what was taught in the first edition of Dr. Ferrier's work, that both are situated in the hippocampal lobule and the lower temporo-sphenoidal convolutions.

Of the localisation of the motor centres we do not propose to speak. With the exception of the observations of Horsley and Schafer with regard to the motor functions of the marginal convolution, nothing of great importance has been added to our knowledge of these centres beyond what Dr. Ferrier taught us ten years ago. We do, indeed, find that our knowledge of these centres is becoming more minute and definite as time advances. While formerly we heard of movements of the muscles of the shoulder, we are now taught that a destructive lesion of a very limited size in a particular part of the cortex causes paralysis of the biceps on the opposite side; and instead of finding that a particular area is related to the movements of the hand, we now hear of a thumb centre, and so on; but in principle and for practical purposes, recent investigations have not added much that is new to our knowledge of the localisation of these centres. This speaks well for the thoroughness with which the work was done by Dr. Ferrier in his first series of experiments.

With regard to the functions of the frontal lobe, Dr. Ferrier finds that electrical stimulation of the area comprising the base of the superior and middle frontal convolutions—the postfrontal area—gives rise to lateral movements of the head and eyes with dilatation of the pupil, while destruction of this area causes paralysis of these movements, resulting in a conjugate deviation of the eyeballs and rotation of the head towards the side of the lesion. The animal however soon regains the power of moving the eyeballs after destruction of the postfrontal regions, and even bilateral destruction of them causes only a temporary paralysis of the movements of the eyeballs and of the lateral movements of the head.

Electrical stimulation of the prefrontal area is not generally followed by definite motor manifestations, although Dr. Ferrier states that in two instances he observed movements of the eyeballs on irritation of these regions. Destructive lesions also seem to show that these regions are functionally related to the movements of the eyeballs and head. In one case of destruction of the postfrontal regions, in which the animal had completely recovered the power of moving the eyeballs and head, subsequent destruction of the prefrontal regions was followed by rapid oscillations of the head, apparent inability to turn the head, except *en masse* with the trunk, and drooping of the right eyelid. Although these symptoms also disappeared three days after the second operation, the author concludes that the "prefrontal regions belong to the same centres as the postfrontal, just as the occipital lobes belong to the visual centres." This conclusion is much strengthened by another line of enquiry. In one case, in which the prefrontal regions were extirpated, and the animal survived the operation for three months, a descending sclerosis was discovered in the innermost bundles of the internal capsule and foot of the crus cerebri on either side, but these degenerated tracts could not be traced beyond the upper part of the pons. In another case the postfrontal centres were first destroyed and seven weeks afterwards the prefrontal regions were removed. Two

months and a half after the second operation a descending degeneration was found in the innermost bundle of the internal capsule and crusta, the bundles most mesially placed showing a less degree of degeneration than those externally placed. This experiment shows that the innermost bundles are connected with the prefrontal region, and those which lie immediately external to them with the postfrontal centres.

These observations afford strong confirmation to the opinion already expressed by the author, that both the prefrontal and postfrontal areas are concerned with the movements of the head, and eyes. Dr. Ferrier finds from his recent as from his earlier experiments, that although extirpation of the prefrontal areas is not followed by any manifest sensory or motor disorder, yet the animals operated upon undergo a decided mental deterioration, and in this conclusion he is borne out by the results obtained by Horsley and Schäfer, and Hitzig and Goltz.

The experimental investigation of the functions of the basal ganglia is surrounded by innumerable difficulties, owing to their complexity, and to the serious injuries which must necessarily be inflicted on other important parts in gaining access to them. The fact that electrical excitation of the corpus striatum gives rise to a tonic spasm of the opposite side of the body, shows that it forms part of the motor nervous mechanism, but Dr. Ferrier rejects Meynert's opinion, that it is a "ganglion of interruption" of the cerebral motor conducting paths. He adopts the view, which is now more commonly received, that this ganglion is an independent centre of the same nature as the motor centres of the cerebral cortex; in his own words, "the corpora striata proper are centres of innervation of the same movements as are differentiated in the cortical motor centres, but of a lower grade of specialisation." Much less is known of the functions of the optic thalamus than even of those of the corpora striata. The facts that irritation of this ganglion is not followed by any motor manifestation, and that when destroyed its fibres undergo an ascending degeneration, warrant the conclusion, that it has some relation to sensory centres and tracts; and it is also probable that the posterior part of it has a special relation to the visual sphere. "It is probable," says Dr. Ferrier, "that the optic thalami, specially related to the sensory tracts, and the corpora striata, specially related to the motor tracts, represent in a subordinate manner all the sensory and motor centres of the cortex, and constitute together a sensori-motor mechanism, subservient to the manifestation of all those forms of activity which do not imply conscious discrimination or true volition."

The author now considers the functions of the hemispheres from a psychological standpoint, and ably discusses such vitally important questions as the connection between brain and mind, the conditions of perception, the feelings and emotions, the appetite and desires, the motives to volition, the growth of volition, the conflict of motives, the acquisition of speech with the related subject of the dissolution of speech or aphasia, the control of

ideation, and attention, with a consideration of the anatomical substratum of this mental act.

A mere enumeration of these subjects shows how impossible it is to enter in this place upon a profitable analysis of Dr. Ferrier's treatment of them. The reasoning is so close that the student of psychology cannot afford to lose sight of a single sentence; and even the busy practitioner will find the masterly manner in which the intricate subject of aphasia is here presented before him of the greatest practical value.

The work concludes with a chapter on cerebral and cranio-cerebral topography, which is improved as compared with the corresponding part of the first edition by the introduction of Reid's admirable diagrams of cranio-cerebral relations.

In bringing our remarks to a close we have no hesitation in saying, that this work is one of which it is scarcely possible to speak too highly. In its first edition it was not simply a treatise on the functions of the brain, but a record of a revolution, effected in great measure by the author's own labours, in one of the highest and most complicated departments of science. And if it is not given to the author to present to his profession and the public so much of a revelation in this as in the former edition, it is not because his right hand has lost any of its cunning, but because medical literature is now permeated with his principles and the medical mind has become saturated with his opinions. To master such a work as this is in itself an education, and whether it be studied with the view of observing what it is possible to achieve by skilfully devised and dexterously executed experiments, of noting the powerful dialectic with which fallacies are exposed and false conclusions demolished, or of following the author through subtle psychological distinctions and abstruse speculations, no one can rise from an attentive perusal of the work without feeling an accession of strength to his intellectual capacity, and being inspired with additional hope for the future of medical science. To recommend Dr. Ferrier's book to the notice of the readers of "BRAIN" would be a singularly superfluous task, and as for the author himself, his reputation is now so widely known and so firmly established that he can afford to feel alike indifferent to adverse or favourable criticism. It may at last be permitted to us to say that no one can pretend to undertake any accurate work in neurology, whether it be for the practical purposes of discriminating obscure diseases at the bedside, or of extending our knowledge of the functions of the nervous system by new researches, without having a copy of Dr. Ferrier's book by his side for frequent reference.

JAMES ROSS.

Leçons sur les Maladies du Système Nerveux, par J. M. CHARCOT: Tome III. 1 vol. 8vo. pp. 518. Paris, 1887.

THIS third volume of Lectures is in every way worthy of its predecessors. We recognise throughout the master's hand, who knows so well how to clothe the wealth of information he has to impart, in the lucid grace of his yet powerful diction.

The first lecture has a pre-eminently historical interest. It consists in an opening address delivered by Prof. Charcot on assuming the new chair of neurology created in the medical Faculty of Paris. For twelve years the founder of the great Salpêtrière School had, by word and deed, laboured to demonstrate the necessity of founding a department for the study of nervous diseases. It required all the influence of his strong will and eminent personality to overcome the multitude of obstacles—the influences of inert officialism on the one hand, of active jealousy on the other—placed in his way. At last the hour of triumph came; and everywhere, outside at least that agonosphere of petty personal interests that surrounds every great enterprise, men of science were unanimous in congratulating Prof. Charcot for his achievement, and the authorities for their wisdom.

We shall endeavour, in the following pages, chiefly to give a brief summary of the contents of this volume. Lecture II. deals with the subject of

Muscular atrophy after articular lesions.—The patient, whose case formed the subject of this address, presented considerable atrophy of the triceps extensor of the thigh, following a blow on the knee. The articulation itself never was the seat of any extensive alterations, though soon after the accident there had been marked loss of motor power. This case is typical of a large class in which arthritic disturbances are followed with atrophy of the extensors. This atrophy is simple, not degenerative, the affected muscles displaying no qualitative alterations in their response to both currents. When their atrophy is considerable they may cease to react; but even then a spark from the static machine often excites a contraction. Prof. Charcot explains the nutritive alteration in the muscle to a reflex paralysis of the motor spinal cells which form its trophic centre. He does not however explain why the arrest of the trophic spinal influence from an *organic*, should give rise to degenerative, from a *functional* cellular disturbance to simple, atrophy. This is a point worthy of further inquiries.

III. *Traumatic contraction.*—It is a well-known fact, that an injury in a subject predisposed to gout, rheumatism, and other constitutional diseases, may give rise to local outbreak of his constitutional malady. The same rule obtains in hysteria. One of the old stagers in the Salpêtrière, whose epileptiform attacks had for some years become less and less frequent, fell one day on a staircase and slightly injured her left ankle. The whole limb was at

once thrown into a state of complete contracture. Prof. Charcot explains this fact by assuming an over-excitability of the motor spinal cells, and shows how similar effects may be observed after hemiplegia, and in certain cases of hysteria where percussion of tendons gives rise to persistent contraction of muscles.

Facial hystero-spasm.—Prof. Charcot describes a curious case of tonic spasm of the facial, with obvious psychical complications, in a hysterical girl. Treatment made things rather worse, until complete isolation was enforced.

IV. and V. *Atrophy and contracture in arthritis.*— Chronic articular rheumatism is divided by Charcot into: (1st) the primitive generalised or progressive type; (2nd) the primitive partial or fixed type; (3rd) Heberden's nodosities. The first usually begins in the metacarpo-phalangeal joints and thence spreads widely. The second remains localised in one or two large joints. The third (often mistaken as of gouty origin) affects chiefly the terminal digital articulations. The morbid-process in all is a dry arthritic nature; and the three types are connected by transitional forms.

A curious feature of the muscular atrophy from joint lesions is that it is often associated with exaggerated tendon-reactions. One even observes in some cases that percussion of the knee gives rise to muscular contractions in distant muscles. This muscular hyperexcitability is related to the contractures, the rigid flexed attitudes, often assumed by a limb affected with painful joint affection; and as Charcot pointed out more than thirty years ago, the singular deviations of bones observed in rheumatic arthritis are due to reflex muscular spasms. Thus both the amyotrophies and the contractures of joint affections depend upon a reflex central nervous disturbance, either of an inhibitory or of an excitatory nature.

Ophthalmic migraine.—In a general paralytic of the characteristic "congestive" type of Falret, each attack was preceded with the appearance of a scintillating scotoma, followed by hemiopia, pain, and vomiting. These symptoms form the "ophthalmic migraine" of French authors, which also includes as subsidiary elements paralytic, aphasic and epileptiform phenomena. At the outset, these fits of migraine were the only symptom of the coming mischief, and Charcot insists upon their grave premonitory character.

VI.–VIII.—These lectures illustrate and discuss several points connected with *hysteria in the male*—a subject which has recently attracted considerable attention in France. Klein, who gives statistics, says that it usually is hereditary and appears after puberty. Boys, however, may also be affected, as Prof. Charcot proves by describing several well-marked instances. Hysterical men are by no means necessarily effeminate; they often belong to the artisan or labouring classes. A case is here described in which a smith developed a characteristic hysterical contracture of the hand after a burn. The details of, and commentary on, the case, are well worth reading in the original. It is to be noted that hysteria in males is occasionally of the severe type, but more frequently is not characterised by violent convulsive or emo-

tional outbursts. Somatic symptoms are often observed without any psychical accompaniments. As a parallel to the smith's case of this "hystérie fruste," Charcot describes that of a girl who likewise presented contracture of the hand after a slight injury, though she never had presented any "hysterical" tendency before that event.

IX. *Spinal disease after injury to sciatic nerve.*—A cabinet-maker, aged 40, received a blow just below the left buttock. From that time he suffered from symptoms of ordinary sciatica, with pressure points, and occasional fulgurating pains and muscular spasms. About three months afterwards the painful symptoms disappeared, but the paralysis and degenerative atrophy of the whole limb above and below the seat of lesion, became more pronounced. The bladder was involved, necessitating daily catheterisations. Incontinence of fæces, and impotence were also observed. Some of the muscles displayed the degenerative reactions; and there was also muscular atrophy in the right leg. Charcot explains these symptoms by assuming a myelitis set up by peripheral irritation.

X. A case of *double sciatica* in a cancerous subject is the theme of some considerations on the frequency and clinical symptoms of the invasion of the vertebræ by cancerous deposits; whilst another, of *cervical pachymeningitis*, is described with special reference to its successful treatment by the cautery, followed by surgical procedures calculated to restore the movements of the rigid lower extremities.

XI. and XII. These lectures contain a full account of, and a discussion on, a very perfect case of *word-blindness*. This form of aphasia in which the patient, though he may still be able to speak and write perfectly, is unable to realise the language value of written symbols, has long been recognised in Germany and England. But in France, where the influence of Broca's discovery of the "speech" centre, and the defective psychological doctrines of the School, stood in the way of further progress in the knowledge of aphasic symptoms, it is only quite recently that the powerful influence of Charcot has broken through the tradition and silenced prejudice. Even Magnan's clear teaching on this subject had scarcely secured a fair hearing. Now "nous avons changé tout cela," and the study of the various forms of aphasia is pursued with much energy and success by a number of French neurologists, supported on the psychological side by Prof. Ribot, and other writers of the same school.

XIII. In marked contrast with the last case stands one of *visual amnesia*, or loss of mental imagery, the description of which is full of interest to the psychologist as well as to the physician. The patient, a highly-gifted and educated man, belonging to the "visual type" of Galton (*i.e.* carrying out his mental processes chiefly by aid of visual representations) suddenly found himself deprived of the power of remembering or recognising objects *as seen*. The reflection of his own face in the glass became unfamiliar to him. Though a fair draughtsman before, he was

unable to visualise, or depict on paper, even simple geometrical figures. He used to be able to learn by heart poetry with the greatest ease, fixing in his memory the image of the pages, and, when reciting, reading off as it were from the recalled sight of these pages. Now all this was changed, and he had to trust to the reviviscence of the words as heard or articulated, that is to say of auditory or motor impressions, in the process of recitation. Strange to say there were but very slight traces of word-blindness; he could read with much the same facility as before—a fact that constitutes a striking exception to the application of the "law of dissolution" to this case. There is a point in the mental condition of the patient which we think deserves to be commended to the attention of psychologists. Though the name of a person or of an object failed to call up the faintest trace of a visual representation, he clearly understood such a word. The explanation of this fact is, we think, to be found only in the theory, that what we call a name is a symbol standing for a complex state of consciousness, involving the associational activity of several or all the special sensory cortical regions, with more or less distinct innervation of the emotional sphere. Hence, even when the leading sense-representation is absent, the remainder of the total conscious process is sufficient for the carrying on of a train of thought. Do we not here have an experimental demonstration of the fact, that the use of abstract or general names is possible without any concurrent visual representation individualising the class which it represents?

The subject of Lecture XIV. is a nosographical revision of *Amyotrophies*, which will be found mentioned in Dr. Tooth's Digest in the present number of BRAIN.

XV. *Tremors* vary as to their rate, as well as to their occurrence, form, and amplitude. Thus in disseminated sclerosis and paralysis agitans they consist of between four and five oscillations a second. The rate of hysterical tremor is six or seven; whilst in alcoholism, mercurialism, general paralysis, and exophthalmic the vibrations are more rapid, eight to nine a second. Among *Choreiform movements* we must distinguish between the meaningless gesticulation of chorea proper, the motor phenomena of athetosis, posthemiplegic chorea, on the one hand, and the more systematized and rhythmical movements of the conditions known as saltatory, malleatory and rhythmical chorea, on the other. These symptoms are most frequently observed along with other hysterical manifestations.

Lectures XVI.–XVII. contain the instructive cases of three children, two brothers and one sister, in whom severe hysteria was determined by taking part in spiritualistic performances—a fact to be commended to the attention of certain dabblers in "psychical research," and more particularly to the author and readers of a recent volume: "Spirit-workers in the home circle; a narrative of psychic phenomena in family daily life."—Charcot takes this opportunity to insist again upon the paramount importance of complete isolation from home and friends in the treatment of hysteria. It is a remarkable fact that, whereas the little patients

had convulsive attacks on meeting one another, or their father and mother, they at once improved when placed among the patients at the Salpêtrière, many of whom are the victims of the graver forms of hystero-epilepsy. There is evidently much exaggeration in the opinion often heard expressed in this country, that cases are "manufactured" by the contagious influence of example, in the Salpêtrière wards. Here we had three young children who developed the disease in the seclusion of their solitary home, and were cured when placed in the very centre of the so-called infection. It is to be remarked that Briquet and Charcot have, long before Weir Mitchell, found the curative value of isolation and moral control, apart from the often unnecessary adjuncts of massage and over-feeding. It is a pity that the latter should have taken such an undue importance in practice as to obscure the efficacy of the former, which are amply sufficient to cope with bad cases of hysteria, even where malnutrition exists. Medical men should at any rate remember that, in some cases, too energetic physical measures neutralise rather than enhance the good effects of the moral factors.

In Lectures XVII.–XIX. (and Appendix) the author returns to the subject of *hysteria in the male*, the relatively frequent occurrence of which is still doubted by many physicians. The reason of this scepticism is that they expect to find in males the mobility which is—erroneously—considered as a pathognomonic sign of the hysterical nature of certain symptoms in women. Hence persistent anæsthesia, a depressed psychical condition, have been considered incompatible with the functional nature of certain nerve troubles often observed in men of strong physique and hard working habits, especially after a shock or a blow. The careful study of the eight new cases now described will convince sceptics that their position requires reconsideration; and the evidence accumulated in the succeeding lectures will no doubt appear irresistible to every impartial reader.

Two cases of traumatic *hysterical brachial monoplegia* are exhaustively described and discussed (XX.–XIX.). The first relates to a cab-driver who a week after a fall on the right shoulder lost the use of his arm. The paralysis was complete; but the muscles, though flaccid, reacted normally to electricity. There was complete anæsthesia of the limb (excepting the fingers and palm), involving every kind of sensation, even that of position in space. The clinical characters and distribution of such an anæsthesia are incompatible with any known organic lesion of the brachial plexus, cord, or brain. Further, ophthalmoscopic examination revealed visual troubles, in the shape of contraction of the field and monocular polyopia. The hysterical nature of the latter was proved by the co-existence of macropia and micropia. A pencil placed close to the eye appeared single and very large; at 8–10 cm. it appeared double; at 15–20 cm. its image was two or three times smaller than normal. The face, as is usual in hysterical cases of this sort, was quite free, and the tendon reactions normal.

In the second case the patient, a bricklayer aged 18, lost the use

of the left arm three days after a fall on the shoulder, and presented a complete anæsthesia of the whole limb. The hysterical nature of the lesion was demonstrated by the coexistence on the left side of hemianalgesia, diminution of special sensation, contracted visual field, monocular polyopia, hysterogenous zones, and other signs. Prof. Charcot very forcibly brings into relief the parallelism between these paralyses, which might be described as caused "by traumatic suggestion," and those obtained in hypnotised subjects by verbal suggestion. The characteristics of both classes are identical, and moreover the distribution of the anæsthesia that accompanies them is the same. The limits of the latter differ from that observed after actual lesion of the plexus, inasmuch as they do not correspond to nerve provinces: they are bounded above and below by planes transverse to the axis of the limb.

The chief practical importance of the study of these cases is to be found in the light thrown upon the phenomena of "railway-spine" or rather of "railway brain," a subject ably discussed in Herbert Page's well-known book. The treatment adopted by Charcot deserves notice In addition to douches and electrisation, he first endeavours to raise the expectant attention of the patient by insisting on the curability of their paralysis. Then by means of active and passive movements, and dynamometric practice repeated several times daily, he endeavours to restore those mental representations of movements, or muscular sense ideas, upon the obliteration of which the inability to move the limb seems to depend. This plan is often crowned with remarkable success, the anæsthesia disappearing *pari passu* with the paralysis.

Further illustrations of functional nervous disorders in the male are given in Lectures XXIII. and XXIV., the occasion of which was a typical case of traumatic *hysterical coxalgia*, a condition well known in England since the masterly description of it given by Brodie. The patient was a sawyer, aged 55, the father of seven children, who never had presented any sign of nerve trouble, until he met with the accident (shock from a steam-engine) that determined the coxalgia. The characteristic system of Brodie's disease is the hyperæsthesia of the *skin* in the neighbourhood of the hip. This was well marked in the patient; in whom also movement or percussion of the bones determined a diffused pain radiating from the hip, followed by a kind of hysterical aura. Moreover the left side of the body was found to be anæsthetic to pricking and temperature, except at the elbow, hip and knee; with partial loss of muscular and special senses. Examination under chloroform confirmed the diagnosis as to the functional nature of this coxalgia of about eight months' standing.

It is a curious and important fact that in hypnotised subjects the suggestion of a fall on the hip produces symptoms exactly corresponding to hysterical coxalgia, including even the well-known referred pain in the knee-joint. We find therein a confirmation of the view, that the mechanism of shock in the production of certain nerve troubles is essentially similar, and depends upon the "traumatic suggestion" already mentioned. Moreover, light massage,

both in the case of traumatic and experimental coxalgia, was followed with temporary anæsthesia and complete flaccid paralysis of the limb.

In Lecture XXV. we find an instructive case of *hystero-traumatic contracture.* The patient, a strong man of 30, butcher by trade, received a blow on the left arm, which became anæsthetic and paralysed without the trace of rigidity. Two days after, he applied, on account of some local swelling, for relief at a surgical clinique, when the limb was put up in a plaster bandage. Six weeks after, on removal of the latter, a contracture had set in, that persisted for several months when the patient came to the Salpêtrière.

Volkmann has described a form of muscular rigidity produced by tight bandaging, and which he explains by assuming that a coagulation of the myosine takes place in consequence of the ischæmia. At any rate electrical examination in such cases reveals alterations in the muscles which also do not relax under the influence of chloroform. In the case shown by Charcot, opposite conditions were observed. That the contracture was of hysterical, and not merely spasmodic, nature was shown by the fact that it was extreme, did not relax at all during sleep, and was accompanied with superficial and deep anæsthesia. Moreover it is easy to produce similar contractures in cases of hysterical subjects with muscular hypertonicity, by applying to the limb a few turns of Esmarch's bandage and so obtain experimentally the very condition observed in the present case. The patient eventually recovered the use of the arm; but the fingers remained flexed at an angle of 90° owing to the supervention of fibroid retraction, a complication not unknown in protracted cases of spasmodic contracture.

The last lecture (XXVI.) deals with *hysterical mutism in males.* One of the patients, whose case is described in the Appendix, has since occupied the attention of the medical profession in this country, where he went under the designation of "the Soho-Sleeper." Hysterical mutism usually follows on an emotion, a fit, or a sore throat; it persists from a few hours to days, weeks, months, or even years, and often disappears suddenly. It is characterised by complete aphonia. The patient cannot whisper, nor even imitate the articulatory movements of the tongue and lips. It is a pure and complete motor aphasia, and something more, as every kind of vocal sound is impossible. The other functions of language are intact. Like other hysterical symptoms it is usually accompanied with some characteristic signs of the neurosis (anæsthesia, &c.) and can be experimentally induced by suggestion, upon hypnotised hysterical subjects.

A. DE WATTEVILLE.

Abstracts of British and Foreign Journals.

Current Nerve Anatomy and Physiology.—Titles and Outline of Contents of Papers which have appeared during the Quarter. By ALEX. HILL, M.D. Cambridge.

COMPARATIVE ANATOMY AND DEVELOPMENT.

1. Central Nervous System of Acephalæ. RAWITZ (*Jenaische Zeitschrift für Naturwissenschaft*, 1887: pp. 384–461, pl. xxv–xxix.)

After a short description of the macroscopic structure of the nervous system of certain mollusca there follows an account of the histological methods used, and then an exhaustive description of the microscopic characters of nerve-cells and nerve-fibres in these animals, illustrated with numerous figures.

2. The Anatomy of the Nervous System in the Gymnophiona. Dr. JULIUS WALDSCHMIDT (*Ibid.* pp. 461–476, pl. xxx–xxxi.)

This is an amplification in certain points of the description of the anatomy of this group of amphibia already published by Wiedersheim. With regard to the remarkable double olfactory nerves first pointed out by Wiedersheim, Walderschmidt gives it as his opinion that the ventral root is the homologue of the olfactory nerve of other vertebrates, and the dorsal root a later secondary formation, depending for its development upon the great relative importance of the sense of smell to these blind animals.

3. The Brain of the Wasp (*Vespa crabro et vulgaris*). H. VIALLANES (*Ann. des Sciences Nat. Zool.*, tome ii. pp. 1–100, pl. 1–6).

The whole of the prebuccal portion of the ganglionic chain is regarded as "brain." This, as in previous articles, the author divides into proto-cerebron, belonging to the first somite, and connected with the eyes; deutero-cerebron, belonging to the second somite, which bears the antennules, and presenting a characteristic

structure due to its "olfactory glomerule;" trito-cerebron, or brain of the antennary segment, which differs in no respects from the ganglia of the ventral chain.

The paper contains a detailed anatomical description of these parts, and also a historical summary.

4. The Development and Innervation of the Nerve-ridge (Nervenhügel) of Urodelan Larvæ. P. MITROPHANOW (*Biol. Centralblatt*, May 1887, pp. 174–176).

5. Man's Structure as Evidence of his History. WIEDERSHEIM (*Berichte der Naturforschenden Gesellschaft zu Freiburg i. B.* 1887; pp. 168–278.)

Includes an account of the characters which distinguish the Nervous System in man from that of other animals, and a discussion of the phylogenetic bearing of these features.

6. The Medullary Folds of the Chick. E. BÉRANECK (*Recueil Zoologique Suisse*, 1887, pp. 305—pl. xiv).

A description of folds and thickenings of the hind brain in the embryo chick, similar to those found in reptiles, and throwing light upon the segmentation of the vertebrate skull.

CORPUS CALLOSUM.

7. The Origin of the Corpus Callosum, a Contribution upon the Cerebral Commissures of the Vertebrata. Part II. H. F. OSBORN (*Morph. Jahrbuch*, pp. 530–543, pl. xxv, 5 *figures in text*).

A continuation of Osborn's work upon the Cerebral Commissures, in which he has proved that they exist in all vertebrates, and are always arranged according to the same fundamental plan. The present paper treats of the Commissures as they are found in certain adult ophidia and aves, and also of their development in the frog and sheep.

8. The Microcephalous Brain of Hoffmann, in which the Corpus Callosum was absent. ONUFROWICZ (*Arch. fur Psychiatrie*, pp. 305–328, pl. viii. and ix.)

Critical summary of all hitherto recorded cases. Anatomical description of this case, in which the Corpus Callosum was completely absent, illustrated with figures showing external appearance

and numerous transverse sections. Except for the absence of Corpus Callosum, Lyra and Olfactory Bulbs, the brain may be regarded as normal although small. The internal capsule was normally developed. Subject was an idiot 37 years old.

CORTEX.

9. The Smell-Centre. A Study in Comparative Anatomy. Prof. ZUCKERKANDL of Graz. (*Publ. F. Enke, Stuttgart*, pp. 124, 7 *lithographs and* 25 *figures*.)

The author gives a description of the typical formation of the cortex-margin in the region of the Cornu Ammonis, and in particular of the callosal convolution (Balkenwindung) first described as a separate structure by himself. He then traces the variations in this formation exhibited in a large number of animals. The comparative anatomy of the olfactory lobe is then traced, and it is shown that when it is reduced in size the lobus Hippocampi suffers a corresponding diminution. The arrangement of the mesial convolutions is then described; the limbic lobe and the inner and outer elements of the arcuate convolution (Randbogen) are brought into a scheme forming three concentric rings completed in front by the roots of the olfactory tract and the variations in the development of the several portions of these rings are described. From the small development of the Cornu Ammonis in anosmatic animals, and especially in the dolphin, it is inferred that this belongs to the smell centre.

10. Illustrations of normal and defective development of the Multipolar Cells of the Cerebral Cortex; of their degeneration in Senile Insanity, and of certain Albuminoid or Protoplasmic exudations commonly found in the neighbourhood of the junction of the white and grey matter of the Convolutions in cases of General Paralysis and ordinary Mania, in which the cases have been more or less acute. EDWARD PARKER, M.D. (*Journal of Mental Science, April*, 1887, pp. 20–25, *with illustrations*).

11. Macro- and Microscopic Investigations on the Nerve-centres. Dr. CASIMIRO MONDINO (*publ. Turin, Unione Tipografico-editrice*, pp. 70, *nine large coloured plates*, 1887).

Devoted to a consideration of the exact structure, connections and mutual relation of the claustrum, nucleus amygdaleus and cortex of the apex of the gyrus hippocampi.

12. A Note on the means of Topographical Diagnosis of Focal Disease affecting the so-called Motor Region of the Cerebral Cortex. V. HORSLEY (*Am. Journ. Mental Science*, 1887, pp. 342–369).

LOCALISATION.

13. The Heat-centre in the Brain. OTT (*Journ. Nerv. and Mental Disease, N. Y.* 1887, pp. 150–162).

14. The Question of Cerebral Localisation, with consideration of Cerebral Disturbances of Vision. REINHARDT (*Arch. für Psychiatrie*, pp. 449–487).

Conclusion of paper which appeared in previous volume.

CEREBELLUM.

15. A Case of Atrophy of the Cerebellum, with Degeneration in the Medulla Oblongata and in the Spinal Cord (probably the result of Alcoholism). Dr. SCHULTZE of Heidelberg (*Virchow's Archiv*, 1887, pp. 331–343).

The case occurred in the person of a man 39 years old. There was a general diminution in size of the cerebellum in all its diameters. Microscopical examination revealed an obvious loss in white fibres; a diminution in the number of cells of Purkinje; atrophy of the corpus dentatum. In the medulla both olives were degenerated. Throughout medulla and cord there were various alterations which are minutely described, but which were not in all respects such as would, according to most theories as to the connections of the cerebellum, be expected. Türck's column and the direct cerebellar tracts were intact. In the upper part of the cervical swelling there was a small patch of degeneration, on the right side, lying just in front of the crossed pyramidal tract.

GENERAL PHYSIOLOGY.

16. Remarks on Evolution and Dissolution of the Nervous System. J. HUGHLINGS-JACKSON, M.D., F.R.C.P., F.R.S. (*Journ. Mental Science*, April 1887, pp. 25–48).

NERVE-FACIAL.

17. The Muscles innervated by the Facial Nerve in a young Gorilla. G. RUGE (*Morph. Jahrbuch*, 1887, pp. 459–530, pl. xxiv.)

This is a study of muscles, although illustrative of the distribution of the facial nerve.

Nerve Auditory.

18. The Question as to the Origin of the Nerve of Hearing, and the physiological meaning of the N. Vestibularis. Professor W. Bechterew (*Neurolog. Centralbl.*, 1887, pp. 193–198).

Presents a summary of the views held by various observers on this matter. The author maintains the opinions as to the connections of the auditory nerve which he published in 1885.

Spinal Cord.

19. The Localized (Punktförmig) Stimulation of the Frog's Spinal Cord. Sirotinin (*Arch. f. Anat. und Physiologie. Phys. Abth.* pp. 154–177, *with Plate II. giving illustrations of the Clamps used*).

The cord was stimulated by puncturing with a needle. The percentage number of times that movement of certain muscles (Ileo-psoas, Semitendinosus Gastrocnemius) resulted from the prick was observed. The region, puncture of which gave rise to the maximum number of movements of one of these muscles regarded as in especial connection with this muscle.

Latent period for contraction of each muscle, after puncture of each region from which it could be obtained, measured.

Cord surrounded by fourth vertebra associated with Ileo-psoas. Fifth vertebra semitendinosus. Sixth gastrocnemius. Comparison of these results with the effects of electrical stimulation.

Reflex Action.

20. A Crossed Reflex in the Frog. O. Langendorff (*Archiv für Anat. und Physiologie. Phys. Abth.* pp. 141–143).

Contains further details with regard to the manner of producing by means of an electric stimulus the crossed reflex already described by L. The reflex movement consists in a tetanic flexion and abduction of the opposite hind leg, when pressure is made with a blunt instrument behind the eye or over the tympanic region.

21. The One-sided and Both-sided Closure of the Eyelid. By the same Author (*Ibid.* pp. 144–147).

22. Contribution to the Study of Reflex Phenomena. The adapted and defensive Reflexes among Mammals from the results of a new experimental method. J. V. Laborde (*Comptes rendus de la Soc. de Biologie*, 1887, p. 65).

After removal of both hemispheres from new-born kittens or guinea-pigs by the water stream (Goltz's method), very extensive appropriate, and co-ordinated reflex actions can be obtained almost up to the time of death.

23. Reflex of the Spinal Cord in the Dog (*Ibid.* p. 110).

Spinal Ganglia.

24. The Physiology of the Spinal Ganglia. Dr. Max Joseph of Berlin (*Neurolog. Centralbl.*, 1887, pp. 172–175).

Experiments made on a number of kittens which were killed six to eight weeks after section of the second cervical nerve, either on the proximal or the distal side of the ganglion.

When the section was made between the cord and the ganglion, the results were just such as obtained by previous observers. When the nerve was cut beyond the ganglion, no change in this structure resulted, but a number of fibres degenerated both in their course through the ganglion and in the posterior root. The larger number of fibres of the posterior root remained intact.

Peripheral Nerves.

25. Termination of Nerves in the Liver. A. B. Macullum, B.A. (*Quart. J. Microsc. Science, March* 1887, pp. 439–460, pl. xxxii.)

Observations made with the chloride of gold method showed that there is an intimate relation between nerve fibrils and liver cells. Nerve filaments enter the cells (frequently dividing in a dendritic manner within their substance), and terminate in bead-like enlargements in the neighbourhood of the nucleus.

26. Some Variations in the arrangement of the Nerves of the Human Body. D. Hepburn (*Journ. Anat. and Physiology*, 1887, pp. 511–514).

27. Note on the Nerve-supply of the Musculus Sternalis. J. C. Lamont (*Ibid.* pp. 514–515).

Nerve Fibres.

28. Histology of crushed (Gequetschen) Nerves. Franz Tangl. (*Arch. f. Mikrosc. Anat.*, pp. 464–470, pl. xxvii). (*Also in Hungarian in Orvosi Hetilap.* 1887, *Nos.* 2, 3, 5, 10 *and* 11.)

Behaviour of the axis cylinder after ligaturing of nerves shows

that it is not fluid, but has considerable powers of resistance and perhaps elasticity.

SYMPATHETIC.

29. Observations upon the persistent effects of Division of the Cervical Sympathetic. P. H. PYE SMITH, M.D., F.R.S. (*Journal of Physiology*, 1887, pp. 25–49).

A series of observations carried on systematically between 1878 and 1884.

CIRCULATION.

30. Physiological Diurnal and Nocturnal Variations in the Brain-pulse. RUMMO and FERRANNINI (*C. R. Acad. des Sciences, Paris* 1887, pp. 310–313).

STAINING.

31. Method of combining Weigert's Hæmatoxyline-copper stain with the use of the Freezing Microtome. D. J. HAMILTON (*J. Anat. and Physiology*, 1887, pp. 444–450).

Gärtner and Wagner on the Cerebral Circulation (*Wr. Med. Wochenschr.* Nos. 19 and 20, 1887).—The authors have gained important results by applying to the problems of the cerebral circulation a method of investigation which has been fruitfully employed in researches concerning the circulation in other organs.

The method consists in measuring the amount of blood which flows through an organ in the unit of time—which therefore passes away by the venous system. In this manner, it is possible to draw conclusions as to the innervation of the vessels of the organ concerned, due regard being allotted to certain other circumstances, especially the blood-pressure.

The authors' experiments were performed on dogs. The flow of blood was registered on a hymograph by means of a cannula placed in one of the external jugular veins, which, in dogs, convey the chief mass of the cerebral blood.

As the observations were limited only to one of the many cerebral venous channels, it was necessary to determine how far that limitation might vitiate the experiments. If an impediment to the outflow from the other exit channels were brought about by an increase in the venous pressure, more blood would flow through the prepared vein, and contrariwise with a sinking of the venous

pressure. But by making the resistance to the outflow in the prepared vein very small in proportion to that which obtained in the remaining effluent vessels, any error arising from the limitation referred to, and from fluctuations in the extra-cranial venous pressure, was reduced to a negligible quantity.

The arterial blood-pressure also had to be taken into account. It is clear that, the calibre of the brain-vessels remaining equal, so much the more blood must stream through them, the higher the pressure under which it circulates. Consequently a fluctuation in the outflow quantity can only be taken as evidence of a change in the calibre of the cerebral vessels, when that fluctuation is not adequately explained by a variation in the arterial blood-pressure.

The influence of the blood-pressure upon the cerebral circulation can be shown by simple experiment; *e.g.* the blood-pressure can be raised by compressing the aorta above the diaphragm. As soon as it be thus raised, the quantity of blood issuing from the cerebral vein increases in an exactly parallel manner to the blood-pressure. This increased outflow continues so long as the raised pressure is maintained, and ceases parallel to the blood-pressure when the compression of the aorta is discontinued. On the other hand, if the blood-pressure be considerably lowered by compression of the ascending vena cava, the outflow quantity immediately sinks; and when the blood-pressure falls under a certain limit, which lies at 30–40 mm. of mercury, the efflux of blood from the brain quite ceases. The same is observed when the pressure is lowered by bleeding.

To control this possible source of error, the arterial blood-pressure was registered throughout all the experiments, by aid of a cannula in one of the crurals.

Certain agents influence very energetically the blood-vessels of various organs through the vaso-motor centre. Such an effect can be brought about in a reflex manner or by toxic means, *e.g.* irritation of a sensory nerve, administration of strychnine. The vessels of the abdominal organs especially are contracted by such means and there occurs in consequence a considerable raising of the blood-pressure. It was of interest to see whether the vessels of the brain also took part in this contraction.

If the cerebral vessels be contracted to the necessary degree, the quantity of blood passing through the brain must diminish, in spite of the increasing blood-pressure, as can be very strikingly shown under like circumstances in the kidney. But the experi-

ments proved that no lessening of the outflow amount took place either by the reflex irritation of the vaso-motor centres, or by the toxæmia of asphyxia or strychnine. In all these cases an increase of the blood-stream was regularly observed, and indeed exactly corresponding to the raised blood-pressure in each case.

This increase of the stream-velocity was particularly marked in strychnine-poisoning.

The fact that no narrowing of the cerebral vessels was produced by the irritation of sensory nerves is of special interest, because it stands opposed to the prevailing theory. It has been supposed that the swooning or convulsions following violent sensory irritation are due to cerebral anæmia brought about by reflex contraction of the brain-vessels. The authors' experiments, with intact skulls, have shown that not only no cerebral anæmia appears in those conditions, but, on the contrary, the brain is more richly permeated by blood, in consequence of the heightened blood-pressure.

Concerning the action of narcotics, the authors found that at the commencement of chloroform inhalation, the circulation in the brain is considerably accelerated, and simultaneously the arterial blood-pressure rises. After a short time, which often is less than a minute, the blood-pressure begins to sink, notwithstanding which the outflow quantity remains increased, and often amounts to three or more times the original when the blood-pressure already has sunk to, or below, the level existing before the inhalation. This acceleration of the blood-stream is thus independent of the blood-pressure, and is only to be explained by a widening of the cerebral vessels. If the chloroform inhalation be continued, the blood-pressure may become so minimal that little or no blood flows through the expanded blood-vessels. If the inhalation be stopped and the blood-pressure thereby be allowed to recover, a copious stream again begins to circulate. If the animal awake from the narcosis, there is often a divergence in the behaviour of the blood-pressure and cerebral blood-stream; while the blood-pressure rises, the flow of blood in the brain diminishes, evidently because the vessels, which had been expanded by the chloroform, contract again to their normal sizes, and thereby neutralise the accelerating influence of the heightened blood-pressure.

No similar property seems to belong to morphia. The fluctuation in the blood-stream which appeared after morphia injection ran an almost parallel course to the variations in the blood-pressure. At first a transient rise of blood-pressure was sometimes observed, and, corresponding thereto, an increase of the outflow; then the

pressure sank below normal, and concurrently the blood-circulation in the brain was slowed—again to be increased as the pressure rose.

Experiments with amyl nitrite produced a manifest expansion of the cerebral vessels, though to a less degree than chloroform.

The effect of direct electrical stimulation of the brain cortex in the motor area was not in harmony with the common theory, which is based upon Kussmaul and Jenner's researches. That theory supposes that an anæmia of the brain, consequent on contraction of the small vessels, exists during an epileptic fit, and that the loss of consciousness is due to the cerebral anæmia.

The constant result of thus directly exciting the cerebral cortex, in the authors' experiments, was a considerable acceleration of the circulation through the brain. This acceleration was usually not observed until 10·30 sec. after the commencement of the stimulation, although the latter, in most cases, was immediately followed by a considerable rise of blood-pressure. At the beginning of excitation there may have been present a slight contraction of the vessels, which delayed the appearance of the quickening of the brain-circulation corresponding to the increased pressure. This point was not fully elucidated by the researches.

The authors further ascertained that the quickened circulation, due to the cerebral irritation, may by no means be caused by the raised blood-pressure alone. It was of longer duration than the latter, and often when the blood-pressure had sunk to the normal or subnormal, the efflux had remained greatly excessive. The acceleration in the blood-stream was quite as distinctly marked in some cases, in which no increase of blood-pressure followed the electric irritation. Thus it was shown that an active hyperæmia of the brain, especially pronounced at the onset of the convulsions, results from electric stimulation of the motor area.

No effect on the cerebral circulation was caused by irritating the divided vago-sympathetics.

Spehl on the Distribution of the Blood circulating within the Encephalon (*Encéphale*, Jan. 1887).—This paper is mainly concerned with a comparison between the amount of blood contained in the head during the wakeful condition with that during chloral hypnotism.

The author indicates, that prior to 1860, the prevailing theory associated sleep with a congested state of the brain. Durham's

experiments, in which he directly observed the vascularity of the brain through a trephine aperture, then showed that the brain during sleep is relatively in an anæmic condition. Many other investigators in later times have confirmed Durham's conclusions, by repeating his mode of research, or by obtaining graphic records of the cerebral movements. On the other hand, an attempt to revive the older theory has not been wanting.

Spehl has sought to elucidate the matter by another process. After subtracting 1 cc. of blood from the carotid of a rabbit, for the preparation of the standard solution, the circulation between the head and trunk of the animal was suddenly arrested by means of an *écraseur* placed close to the head, and the latter was finally separated without the loss of any blood. The total amount of blood contained in the head, and that in the remainder of the animal, were then separately determined.

The mean of five experiments performed upon rabbits in the wakeful state gave $\frac{1}{8}$ as the relative amount of blood contained in the head to the total mass of the blood; whereas the mean of six estimates in the case of sleeping rabbits showed that the head contained only $\frac{1}{11.5}$ of the total blood.

In each series of experiments the mean weight of the bloodless head was $\frac{1}{10}$ that of the exsanguineous animal.

The author thus summarises his conclusions :—

1. During the sleep produced by chloral, the brain, taken, *en masse*, is anæmic.

2. Chloral hypnotism very closely resembling natural sleep (Labbée, Gubler, Rabuteau, Claude Bernard, Liebreich, Bouchut, &c.), it is probable that during the latter the brain is equally anæmic.

3. During sleep, natural or otherwise, certain parts of the brain are in activity, while others are in repose.

4. The active portions probably are congested; the parts in repose are physiologically anæmic.

ERNEST BIRT.

Zelgersma on the Morphogenesis of the Crus Cerebri (*Nederl. Tijdschrift voor Geneeskunde*, No. 21, 1887).—During the anatomical and microscopical examination of five cases of idiotcy, the author was struck by the great influence which the degree of development of the great hemispheres in idiots has on the evolution of the crus cerebri. A primary severe lesion of the cortex cerebri in the fœtal period or in earliest childhood,

existing during a long time, and causing secondary degeneration and inhibition of evolution in certain parts in the crus cerebri and medulla oblongata, characterised these five cases. In three cases hemiatrophia cerebri was demonstrated; in two other cases the alteration of the crus was very evident when compared with normal preparations. The alterations in all cases were nearly the same. They were as follows:

Atrophy of pons varolii and nuclei arciformes; in cases of hemiatrophia cerebri, the cells as well as the transverse bundles and direct fibres to the cortex, were affected on the same side. The pyramidal system was not always affected; here atrophy was only evident when the lesion of the cortex was severe and extensive. The author never saw degeneration of pyramids extend beyond the reflex centres in the medulla oblongata or spinal cord. Besides the atrophy of the pyramidal system, there was atrophy of the transverse fibres in the corpora pyramidalia near the nucleus olivaris in four cases. In all cases the transverse fibres of the pons varolii, which pass through the raphe and brachium pontis, to the cerebellar hemispheres of opposite sides, were atrophic. The nuclei arciformes, cells as well as nerve fibres, were affected. Bechterew's nucleus reticularis, as well as Meynert's bundle of fibres were atrophic. Alteration of nucleus olivaris was not constant. In four of the cases a bundle of fibres from the cerebrum to the nucleus olivaris, described by the author on a former occasion, was always atrophic. In one of the cases the tract between the olive and cerebellum through the raphe and corpus restiforme of the opposite side was atrophic. In four of the cases the cerebellar hemispheres on the opposite side to the brain were atrophic. In one case, the nucleus dentatus also was affected. In all cases the pedunculi cerebelli ad cerebrum were atrophic.

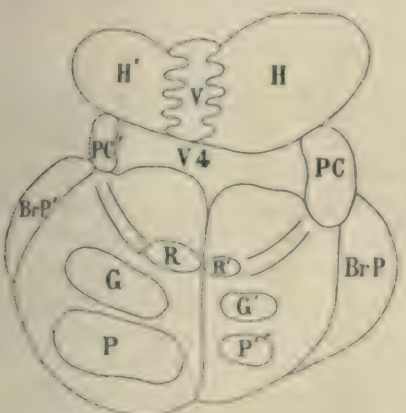
A transverse section through the pons varolii behind the posterior corpora quadrigemina shows a characteristic asymmetrical configuration, in consequence of the passage of decussated and direct, atrophic and normal fibres; this condition is represented by the diagram on page 287.

The nucleus ruber, substantia nigra, and corpus Luysii, are all atrophic. The alteration in the optic thalamus is very complicated. The anterior part is generally atrophic.

There was no affection of the corpora geniculata interna and externa, corpora quadrigemina anteriora and posteriora, and the pulvinar.

From this description, the author concludes the existence of two

different systems; one which connects the great hemispheres with the cerebellum, and the other which connects the hemispheres with the reflex arc (system of pyramids and fillet). The first connection passes through the capsula externa, pes pedunculi, pons varolii, nucleus olivaris (decussating in the raphe), brachium pontis and corpus restiforme to the cerebellum; the fibres from the pons go exclusively to the cerebellar hemispheres; those from the nucleus olivaris pass to the vermis cerebelli back. From the cortex cerebelli the path returns through the nucleus dentatus, pedunculi cerebelli ad cerebrum (decussating near the corpora quadrigemina), and the nucleus ruber, from which radiations arise to the capsula interna and optic thalamus. The difference between degeneration in this system, to which the author gives the name of



P. Pyramids.
G'. Ganglia of Pons Varolii.
R. Nucl. Reticul. pontis.
BrP. Brach. Pontis.
PC. Pedunc. cerebell. sup.
H. Cerebell. hemisphere.
V. Vermis cerebelli.
V4. Ventric. quartus.

intellectual path, and degeneration in the pyramidal and fillet system, is that the former is not governed by the Wallerian law. All these groups of cells which are found in the intellectual path are always degenerated. In the pyramidal and fillet system the degeneration ceases at the first group of cells which is met with. The author concludes that the above-mentioned ganglia are dependent on the evolution of the "intellectuorium."

These results induce the author to suppose, that there exist in the medulla oblongata and crus cerebri three systems of nervous elements: (1) the above-described intellectual tract; (2) the connection between the "intellectuorium" and the reflex arc; and (3) the reflex arc.

This supposition the author proves to be strongly supported by the history of the development of the nervous system. The

function of his "intellectual tract," the author supposes to be of a psychological nature.

The author discusses in three chapters the properties of these systems, and refers the reader for further information to a more elaborate paper in the course of preparation.

J. A. VOORTHUIS, M.D. (Amsterdam).

// BRAIN.

JANUARY, 1888.

BRAIN.

JANUARY, 1888.

Original Articles.

MUSCULAR HYPERTONICITY IN PARALYSIS.[1]

BY A. HUGHES BENNETT, M.D.

HAVING been requested by the Council of the Neurological Society to provide a subject for consideration at the present meeting, I have selected one, which will require no advocacy on my part to ensure an interesting and profitable discussion. The morbid condition which will form the basis of this communication is of sufficiently frequent occurrence to render its study instructive to the practical physician, and the explanations of its phenomena are so contradictory and obscure, that their investigation will prove equally attractive to the speculative pathologist. A problem which is at the same time practical and scientific, which almost daily comes before us only to baffle our efforts at solution, is obviously well-adapted for the deliberation of a congress of medical experts. My duty in opening the debate will simply be to lay the case before you, to indicate the difficulties of the position, and to invite discussion thereupon. I trust that those who follow me will, in some measure, illuminate the darkness into which I am about to grope my way, not without feelings of considerable insecurity.

The general subject which it has been suggested I should lay before you is the apparently paradoxical condition of increased muscular tonicity as associated with motor weakness —a question in its entirety so vast, that it would be hopeless

[1] Paper read before the Neurological Society of London, on July 7, 1887.

for me to attempt to cover the whole ground in the time at my disposal. I will therefore limit my observations to certain special points, that will serve both to direct attention into channels convenient for discussion, and to provide a text for those desirous of dealing with the subject in a more comprehensive manner.

I propose first, to illustrate the association of muscular hypertonicity with motor weakness, by the brief description of a morbid state, which although commonly met with in practice, is apparently neither generally recognised nor understood, and which moreover is not formulated in any of our text-books on medicine; and secondly, to hazard such explanations of its phenomena as seem warranted by the present state of scientific knowledge.

An individual in his or her usual health and without discoverable cause, becomes aware of a feeling of weakness in one or both lower extremities. This at first is so slight as to attract little attention. As months pass by, very insidiously the debility becomes more marked. This condition may continue with little change for many months, or even years, nevertheless it very slowly increases. The sufferers consult many doctors, from whom they derive little benefit or consolation. If a woman, she is probably pronounced to be hysterical; if a man, he is hypochondriacal: or an explanation in his case is abandoned in despair. In both instances a fertile opening for plunder is provided for the bone-setter and charlatan. When seen at this stage, that is, from a few months to even several years after the first discovery of the weakness, the patient still may preserve robust general health. The most careful and exhaustive physical examination fails to discover anything objectively abnormal, except evidence, 1st, of motor weakness, and 2nd, of excessive contraction of the muscles to percussion of their mass or tendons.

On investigating the first point, movement is seldom found to be completely lost, the actions of the affected parts can usually be performed in a natural manner, but are simply deficient in power. There is no special peculiarity of gait, it is only weak, shuffling, and wanting in vigour; the limbs feel heavy, and the patient is easily fatigued by efforts at

walking. There is special difficulty in ascending stairs, and even on level ground the feet are liable to trip over any irregularities on the floor. The points and inside of the boots are worn. The same condition may exist in the upper extremities, the movements being natural in kind, but deficient in quantity. The patient soon becomes tired when writing, sewing, and in the performance of accustomed acts. All the finer actions are slow and clumsy. The grasp of the hand, as determined by the dynamometer, is weak. Movement whenever modified is simply defective in vigour, without cramp, spasm, pain, inco-ordination, or other qualitative peculiarities.

The second prominent feature in these cases is the behaviour of the muscles of the affected parts when percussed. The phenomena which in health are observed to follow the usual test of a blow on their substance or tendon, are exalted in degree, although apparently remaining the same in kind. Expressed clinically, the response of the muscle to percussion is increased, and the tendon phenomena are exaggerated. Every degree of exalted action may exist, from the slightest elevation, impossible to differentiate from health, to the most extreme demonstrations of muscular excitability. In advanced cases trepidations and cloni may be produced in various situations, especially at the ankle and knee. These, however, are usually absent or only imperfectly developed. Tapping the bone in its neighbourhood is often sufficient to cause contraction of a muscle.

With the exception of these two particulars, namely, motor weakness and muscular irritability to percussion, nothing further abnormal is to be found in this class of case. There is no marked rigidity or spasm, although sometimes there seems to be a feeling of somewhat increased muscular tension to passive movement. The superficial reflexes are usually normal; if anything, slightly increased. Sensibility is everywhere unimpaired, and no pain accompanies the complaint. Occasionally the patients assert they experience a feeling of numbness or formication; but this is entirely subjective, and the most careful testing fails to determine any alteration of sensibility. The so-called muscular sense is generally intact, though in some instances I have found

slight loss of capacity to localise movements with the eyes shut. In these cases, however, some doubt existed as to the uncomplicated nature of the disorder. There are nowhere primary trophic changes of any kind. In very chronic cases the affected limbs become somewhat smaller than the others, evidently due to physiological atrophy from diminished functional activity, as the decrease in size is universal, and the electrical reactions remain normal. The affected parts are cold, and sometimes slightly cyanotic, but not to any great extent. Intellect and the various organs and functions of the body are normal, the bladder, rectum, and sexual apparatus remaining as in health. Occasionally, but rarely, the patient complains of some difficulty in retaining urine for long periods, otherwise the bladder and its accessories are intact.

The foregoing brief account may be looked upon as typical of the group of symptoms under consideration, and serve to describe the majority of cases, as they have come under my observation. The only complaint made by the patient is motor weakness, and the only objective sign discoverable by the physician is excessive muscular contraction on percussion. As in other morbid conditions, there are varieties in severity, course, complications, termination, and so on, which deviate from the more common forms. For example, as to distribution, the most ordinary seat of the affection is the lower extremities. Usually it begins in one leg, and sometimes but rarely remains limited to it. A young lady, aged 26, and otherwise in good health, suffered from the characteristic symptoms of this complaint, and limited to the left lower extremity. This appeared very gradually, remained for three years, and slowly disappeared. She is now perfectly well. As a rule, however, the morbid state after a shorter or longer interval extends to the other leg, thus constituting the paraplegic form of the disease, one extremity remaining more advanced than the other. This is by far the most common variety. Beginning in the leg, the malady not infrequently spreads to the arm of the same side, producing the hemiplegic type. Occasionally the weakness and muscular irritability to percussion is limited to one side of the body, more frequently

however, the other soon also becomes affected, even if to a lesser extent. Less commonly all four limbs are equally involved, and still more rarely every muscle in the body shows signs of instability. For example, in 'BRAIN,' for July 1886, I described a case of a young woman, aged 26, who for two years suffered from general motor weakness. All her muscles displayed extreme irritability to percussion, and clonus could be produced by tapping the tendon of any of them, including those of the face, neck, and trunk. This girl subsequently recovered.

The condition I am describing is met with in every degree of severity. From the slightest diminution of physical power and exaltation of muscular irritability to percussion, to complete paralysis with extreme excitability—as evidenced by the production of cramps, spasms, trepidations, and cloni on the slightest provocation—every possible variety and degree may exist. In the cases under the first category it may be difficult to determine if the actual condition found is morbid or not, as the muscular response to percussion in health greatly varies. In those under the last there is always a doubt, unless the case has been followed from the beginning, whether the symptoms are not the result of secondary degenerations from a gross lesion higher up in the pyramidal tract.

Again, the subjects of this disorder may be liable to various symptoms not of necessity associated with, or essential to it, and which may sometimes complicate the diagnosis. Aching in the back and limbs, subjective sensations of numbness, headache, depression of spirits, and so on, may be present, as in any other disease. As a rule, all these symptoms are conspicuous by their absence.

In most cases the onset of the complaint is very insidious, and the progress is very slow and chronic. For example, I have a man under observation who has suffered from this affection for 8 or 10 years, and without any perceptible change either for better or worse. In others, however, with apparently identical symptoms the onset has been very sudden, and after a rapid course the symptoms have disappeared with equal promptness. Thus a young man, aged 27, while in his usual good health, with no apparent cause became weak in his legs,

This increased so rapidly that in a week he could scarcely walk; the debility extended to his arms. On examination, the fact of the motor weakness was established; there was, in addition, extreme readiness of the muscles of all four extremities to contract on percussion, and clonus, more especially of the ankle and knee, could easily be provoked. Beyond this absolutely nothing abnormal could be detected, and there was no rigidity of the muscles. This man completely recovered in a few weeks, with rest and large doses of chloral hydrate, and he has remained quite well ever since. Between these two extremes, namely, those who seem to remain for years in statu quo, and those whose illness is completed in a few weeks, are many who run a medium course of varying periods and duration, ending in more or less complete recovery.

In my experience the two sexes are about equally subject to this disorder. Of the last twenty uncomplicated cases, twelve were males and eight females. The ages in my cases have ranged between twenty and forty years. I have not been able to determine any cause for the complaint, either predisposing or exciting. In one instance only, two brothers were affected; in none of the others was there any special evidence of hereditary predisposition to this or other nervous disease. Some of the patients were subject to exposure and hard physical work, but quite as many had favourable surroundings and were of sedentary habits. No special association with syphilis or other constitutional peculiarity could be determined.

The final evolution of this group of symptoms is difficult to ascertain in a sufficiently large number of cases to enable me to speak with certainty on the subject. Owing to the very chronic nature of their complaint, many patients are lost sight of. At the same time the cases which I have had opportunities of observing for some years have enabled me to formulate the following conclusions: 1st, some of the patients completely recover; 2nd, others continue for many years without apparent change; and 3rd, others slowly progress, the disease extending in distribution from below upwards, and increasing in severity. On each of these heads a few details may be given. At the onset it may be observed that in the cases composing these three groups the symptoms are apparently identical, and

there is nothing to give any indication for making a prognosis.

Complete recovery may take place, sometimes rapidly, but more commonly very slowly. The young man cured by rest and chloral, already referred to, is an instance of the former. The girl already mentioned, who had been ill for three years, is another example. Improvement takes place, as a rule, more slowly. Thus the young lady, of whom I have before spoken as having been affected in one leg only, began to recover six months after she came under observation. Slow improvement continued for three years, after which she expressed herself as quite well. No special treatment had been employed, and the favourable termination has since been maintained. Again, an omnibus conductor, aged 25, had the typical paraplegic variety of the affection. Treatment of various kinds for many months had no appreciable results. For two or three years his condition remained unchanged. Recently, however, he presented himself perfectly well, having gradually improved during the last six months, and without treatment of any kind except that of rest. I could cite several other instances of the same kind. Not unfrequently partial recovery only takes place. In these cases considerable power returns, but not to the full extent. Although the motor activity improves, the muscular irritability to percussion does not diminish in a corresponding ratio, but usually still remains abnormally exalted.

The second class is that of persons who, while apparently in exactly similar circumstances as those just considered, remain for years without undergoing perceptible change in their condition. I have watched several of these for between five and six years. The weakness and muscular irritability to percussion continue as before, but there is no tendency to the development of new complications, or even to an increase of the existing state. The patients have for the most part abandoned medical treatment, and have so far become reconciled to their infirmity as to do the best they can under the circumstances. I recently saw a man whose case I carefully investigated, and minutely recorded just five years ago. He stated that his general health was excellent, and that he was

not conscious of the slightest change in his condition having taken place since he was first examined. On going over my notes, I was unable to discover anything which deviated in the smallest particular from my former report.

The third group of patients are those who seem gradually to get worse. The advance is, as a rule, very slow in progress. The morbid condition extends from one limb to the other, till at last the entire body is affected. The severity of the symptoms also increases, but in my experience not to any great extent. It is probable, however, that we lose sight of the worst cases among our hospital patients, who usually gravitate into the infirmaries. I have myself never seen a case such as has just been described, afterwards pass into one of typical spastic paralysis with its characteristic rigidity of muscle. I am not prepared to affirm that this does not occur. I can only assert, that I have never seen it in any of those cases which I have had an opportunity of watching for five or six years.

On the subject of treatment I have little to say. Rest is undoubtedly an important element, as exertion or fatigue increases the severity of the symptoms. Hydrate of chloral in large doses temporarily depresses the hyper-excitability of the muscles, but whether it induces any permanent beneficial result I do not know. In one case, and the worst I ever saw, the patient after having been afflicted for three years with this illness, recovered in a fortnight after an attack of acute pleurisy. All the usual therapeutic measures, massage, electricity, blisters, actual cautery, setons, &c., have in my experience been followed by no appreciable results. Those who have recovered have done so in the progress of time, and I have been unable to give the credit to any particular remedy.

The group of symptoms which I have described is, as has been already stated, commonly met with in every rank and condition of life. Its existence is frequently overlooked, its importance underrated, and its nature misunderstood by the practitioner. Its consequences, however, very seriously impair the comfort and professional capacity of the patient. It is therefore highly important, not only that the morbid symptoms be recognised and studied with the attention their gravity

merits, but that if possible some estimate be arrived at as to their true significance. Recognising the facts, and admitting the existence of a malady in which motor weakness and abnormal irritability of muscle to percussion are the characteristic and only discoverable features, we have to enquire what are the causes, pathology, prognosis, and treatment of this clinical condition. In attempting to solve these difficult problems, the ground may at once be cleared by the admission that the manifestations of this disorder are strictly limited to the muscles, and motor elements of the nervous system. Every other tissue and function of the body appears to be unaffected. There is no evidence that the muscular substance itself is the source of the disease, as its structure and nutrition are apparently normal, and its alteration in function is not of that nature which would occur from primary changes in its tissues. The seat of the morbid process therefore narrows itself to the nervous apparatus that presides over muscular contractility. Of the morbid anatomy we know nothing. In seeking an explanation of the phenomena we are therefore compelled to enter into the field of speculation, and to utilise collateral physiological and pathological knowledge in attempts to throw light on the subject. Hence there is ample scope for exercising the imagination and ingenuity of the scientific pathologist, and a wide field for divergence of opinion and argument. Discussion will be facilitated by the acceptance of certain elementary physiological premises. Before the audience I now address, it is unnecessary to enumerate these in detail. I therefore confine myself to briefly formulating such general propositions connected with the matter in hand, as in my judgment our present state of knowledge warrants, and concerning which there is a tolerable consensus of opinion.

I. Healthy muscle contracts when its fibres are subjected to sudden stretching (extensile stimulus). This may be induced by percussion on its substance, and still more effectively by a blow on its tendon, the last constituting the so-called tendon phenomenon. The mechanical condition necessary for the production of the response following percussion, is sufficient passive tension of the muscular fibre.

II. This property is intimately associated with and obeys the same laws as what has been termed reflex muscular tonicity, or that state of slight constant tension which is the characteristic of healthy living muscle.

- A. Both are *abolished* when there is interruption at any portion of the reflex arc of which muscle is the terminus.
  - (*a.*) At the posterior spinal roots or sensory nerves, as in locomotor ataxy or neuritis.
  - (*b.*) At the ganglion motor cells, as in poliomyelitis.
  - (*c.*) At the anterior spinal roots or motor nerves, as by experiment or injury to nerve.
- B. Both are *increased* by causes which augment the excitability of the same reflex mechanism. The circumstances under which this takes place are—
  - (*a.*) Idiopathic excitability of reflex centres of cord.
    1. Isolated, as in strychnine poisoning.
    2. Associated, as in neurasthenia.
  - (*b.*) Excitability of reflex centres, secondary to
    1. Direct influence from afferent and efferent elements.
       - (*a.*) Afferent (reflex), as from cutaneous or visceral irritation.
       - (*b.*) Efferent (exhaustion), as from fatigue or over-exhaustion.
    2. Indirect influence, as result of severance from pyramidal tract. This is variously explained by—
       - (*a.*) Irritation of sclerosed lateral columns.
       - (*b.*) Removal of cerebral inhibition.
       - (*c.*) Unbalanced cerebellar action.
       - (*d.*) Nutritive change in ganglia due to severance from trophic centres of cortex.

III. The preceding considerations show that integrity of the reflex arc is essential for the maintenance of muscular tonicity, and the response of muscle to extensile stimulus, or percussion. It is probable that both these phenomena have the same physiological significance, and that the one is dependent on, if it is not an identical process with, the other.

Throughout this paper, in using the terms muscular irritability or excitability, it is to be understood that those expressions are employed only in reference to this tonicity, or property of contracting under the stimulus of percussion.

Admitting the soundness of the preceding general propositions, assuming that increased response of muscle to percussion or extensile stimulus is due to hypertonicity, and that the essential factor for its production is an exaggerated excitability of the ganglion cells of the cord, it has next to be determined more particularly under what circumstances the latter change takes place. For descriptive purposes these may be considered under three heads. 1st, irritation arising primarily in the ganglion cells themselves. 2nd, their irritation due to changes in their afferent and efferent elements. And 3rd, their irritation secondary to influences acting through the pyramidal tracts.

The spinal centres may be primarily rendered unstable in many ways. Strychnine, alcohol, and other poisons, for example, may directly damage their nutrition and produce temporary hyper-excitability of their function. Changes in the quality of the blood, such as anæmia, fevers, acute disorders, &c., may in a lesser degree be followed by the same results; and it is common in such circumstances to find muscular weakness, increased tendon phenomena, and a tendency to cramp and stiffness in the muscles. The same irritability is present in circumstances in which the general system is in a state of subnormal nutrition, as it seems to be a neurological law that nervous depression is associated with instability of function, excessive motor display, and an abnormal readiness to respond to stimulation. Hence in childhood, where the nervous system is as yet imperfectly developed, the characteristic restlessness and proneness to muscular demonstration on the slightest provocation. The diseases at this period of life are also of the same type, as seen in chorea, tetany, convulsions, &c.; and these, as is well known, are usually induced by debilitating conditions, such as syphilis, rickets, fright, and so on. In the adult the same holds good, if to a lesser extent, and loss of blood, fatigue, diarrhœa, cholera, sexual excess, excessive discharges, and other ex-

hausting maladies, may cause temporary cramps, spasms, and other evidence of muscular hyper-excitability. In chronic diseases such as phthisis, it is quite common to find patients in whom the muscles react with abnormal activity to percussion, and in whom the tendon phenomena are exaggerated. In patients convalescent from various diseases I have often found the same, which symptoms disappeared when health was completely restored. In all of these cases the grey matter of the cord participates in the general debility and malnutrition of the tissues of the body, and as a consequence it is in a state of morbid excitability, which manifests itself by abnormal muscular hypertonicity. The same condition is still further, and permanently developed, in those states of nervous habit characterised as the neurotic diathesis, in some of the varieties of which, such as hysteria, this muscular peculiarity often reaches a high degree.

In addition to direct primary irritation of the reflex centres themselves, these ganglia may be thrown into a state of over-activity as a result of morbid impressions derived from their afferent or efferent elements. The well-known experiments of Brown-Sequard demonstrate that injury to *sensory* nerves may induce so-called spinal epilepsy: in other words, a chronic state of extreme hyper-excitability of the grey matter of the cord.

The same has been abundantly proved to be the result of various clinical conditions, and may follow wounds, and accompanying cutaneous, visceral, joint, and other affections, constituting the so-called reflex hyper-excitability of the cord. Whether the stimulation of the peripheral afferent elements induces central changes by direct irritation, by reflex circulatory alterations, by simple functional exhaustion, or by an ascending neuritis, need not now be discussed. The fact remains, that irritation of sensory nerves in any part of the body may conduce to over-activity of the motor cells in the cord to which they are attached. The permanence and degree of the peripheral stimulus appears to bear some relation to the amount and distribution of the reflex effects produced. These, in consequence, may be local or general. If the excitant be moderate, the result, by irritation, is over-action; if excessive, by exhaustion, is abolition of function. Hence

hyper-excitability of motion on the one hand, and paralysis on the other. Thus in the same nervous mechanism, and from the same cause, may arise both irritability and incapacity of muscular action, or as it has been called, a state of "irritable weakness," the two phenomena being essentially the same in kind, only differing in degree. In this way irritations originating in the uterus, kidneys, bladder, prostate, teeth, and other localities may, according to their permanence and severity, cause either spasm or paralysis, or both. In connection with this subject one point may be noted, that a peripheral irritation on one side of the body may not only induce reflex motor responses on the same side, and on the same segment of the cord, but on the other side and in various other regions of the body. This shows that the excitation induced by the afferent stimulus has extended through the grey matter of the cord itself, and is therefore the result of an active irritation process propagated from cell to cell. The effects produced will vary in intensity and distribution according to the character of the excitement, and the original stability of the central nerve elements. Thus the effect of a decayed tooth in one man is slight and local, in another is severe and general. In some constitutions a wound is innocuous, in others it is followed by tetanus.

With regard to the influence of the *motor* elements on the central reflex centre, clinical evidence appears to show that fatigue, muscular exertion, and over-work, lead to instability of the ganglionic cells. This is notably instanced in the case of professional hyperkinesis, or so-called cramp. Excessive physical labour has for long been supposed to have originated various forms of spinal disorder. Dr. Weir Mitchell and Dr. Donkin have recently advanced reasons to indicate that over-fatigue is the chief exciting cause of spastic paralysis, with its well-marked hypertonicity of muscle.

The preceding considerations would seem to indicate, that peripheral irritation or exhaustion of both afferent and efferent fibres may alike lead to secondary hyper-activity of the central ganglia with which they are connected, although the last may have originally been in a healthy condition

Besides the artificial or constitutional circumstances which

directly bring about primary instability of the spinal nerve centres, and the irritation of their ingoing or outgoing paths which produce the same as a secondary consequence, a like pathological condition may follow a totally different source of origin, which brings us to the consideration of the third series of conditions under which hyper-excitability of ganglion cells may be produced, namely, through the influence of the pyramidal tract and cortex of the brain. Although the reflex mechanism already described is the essential factor in the production and maintenance of muscular tonicity, its action is presided over and controlled by influences from above. In health there is a state of equilibrium between the higher and lower centres which permits the normal amount of muscular tone, but if the former are in any way interfered with, the latter become deranged in a corresponding manner. By means of the pyramidal tract, the spinal centres are in direct communication with certain areas in the cortex cerebri. Experimental irritation of the Rolandic regions causes muscular spasm, showing that a stimulus applied to the cortex is by the pyramidal tract conveyed to the spinal ganglia, and through them to the muscles. Irritation by disease of the same regions produces the same effects, and during the paroxysms of muscular convulsion, which as a consequence of the cortical excitement ensues, I have often satisfied myself that in addition to the spasm, the tonicity of muscle was augmented as evidenced by an increase of the knee-jerk. In such cases the exalted functional activity of the lower ganglia may remain, even after voluntary motion has been abolished, owing to the temporary exhaustion which follows an explosion of the cortical centres. When the secondary irritation of the cord subsides, this symptom quickly disappears. The same condition is met with in the so-called "late rigidity" following certain brain lesions. Irritation of any portion of the pyramidal tract may in a like manner induce tonic spasm, and if the cause be chronic the effect may be persistent. The excitability is directly conveyed to the ganglion cells of the cord, and induces in them that exaltation of function which causes excess of muscular tonicity.

A remarkable and seemingly paradoxical fact is that

destruction of the same regions, namely, the motor cortical centres and the pyramidal tract, is gradually followed, not immediately but after an interval of time, by the same hyper-excitability of the spinal centres. This is notably seen in the late rigidity succeeding brain lesions and in primary lateral sclerosis. The explanation of this phenomenon will be afterwards discussed; it is sufficient at present, in connection with this fact, to state that the cells in the cerebral cortex are the trophic centres of the fibres in the pyramidal tract, and that when the former are destroyed the latter in consequence undergo degeneration as far as the ganglia in the cord, which, although left structurally intact, develop as a result hyper-excitability of function.

To repeat and sum up, exaggerated activity of the ganglionic cells in the grey matter of the cord which results in hyper-tonicity of muscle may result from—1st, primary irritability of the cells themselves (strychnine, &c.); 2nd, irritability of the entire reflex arc in common with the whole nervous system (neurasthenia, &c.); 3rd, irritability of cells secondary to abnormal state of their afferent and efferent elements (injuries to sensory or motor nerves, &c.); 4th, direct irritation of cortex cerebri or pyramidal tract (tumour of brain, early stages of lateral sclerosis, &c.); and 5th, destruction of cortex or pyramidal tract (cerebral softening, and late stages of lateral sclerosis, &c.). The essential result from all these causes seems to be the same, varying in intensity and distribution according to the severity and extent of the existing irritation or destruction. An important distinction as to the *time* of production between the two last is, that in the former the effects are immediate, and within certain limits are proportionate to the amount of irritation; in the latter the effects are gradually developed, and depend upon the situation rather than the severity of the irritation.

These, as far as we know them, are the chief phenomena concerned in the production of hypertonicity of muscle. There remain to be enumerated the hypotheses which have been advanced to explain the facts. The more important only of them will be referred to.

The theory of the French school, as especially represented

by Bouchard, Charcot, and Brissaud, maintains that the symptoms of hypertonicity present in spastic paralysis are due to an irritative process, produced in the ganglion cells by the degenerated contiguous fibres of the lateral columns. That this hypothesis is plausible cannot be denied, and it is probable that it is a factor in the production of the clinical symptoms in question. It is, however, as thus expressed, an incomplete explanation, and not the essential cause of the phenomena, as hypertonicity of muscle may exist in its most typical form and in an extreme degree when no degeneration of nerve fibres can be demonstrated, as in various so-called functional complaints, such as hysteria. Moreover, it may occur with such rapidity, and under such circumstances, as to preclude the existence of gross structural alteration of tissue, as in strychnine poisoning and the hypnotic state.

Another theory, and perhaps the most popular one, assumes that the increased excitability of the cord centres which leads to hypertonicity of muscle is the result of simple severance of the cerebral from the spinal centres, by which the controlling influence of the former is removed from the latter. By some it is supposed that the cortex cerebri actively inhibits the lower ganglia, while others consider that the actions of the last are only as it were "let go" by the removal of a higher passively controlling force. It may be admitted that in the absence of brain influence the reflex excitability of the cord is augmented, and, on the other hand, that volitional efforts in a certain measure modify its manifestations, but whether the above explanations are sufficient to fully account for these facts is open to doubt. If the cerebral centres, by an active process, inhibited, in the strict sense of the term, the centres concerned with muscular tonicity, we should expect to find that when these were irritated, the muscle would relax. Irritation of the vagus directly controls, and if persisted in arrests, the action of the heart; hence it may justly be called an inhibitory nerve. No such effect follows stimulation of the cerebral centres; on the contrary, as has already been stated, during excitement or explosive discharge from the motor cortex, as takes place in those cases of epileptiform convulsion without paralysis, which points to irritability and not de-

struction of nervous tissue, the tonicity of muscle is not diminished, but is actually increased. Experimental stimulation of the optic lobes in frogs controlling reflex acts is probably only another form of peripheral sensory irritation, and does not prove the existence of a special inhibitory centre. That mental effort modifies the effects of muscular tonicity is mainly due to the putting in action of antagonistic muscles. It does not control the organic reflexes, but only those in which voluntary muscles play a part. The "let go" theory, on the other hand, does not account for the fact that in sudden destructive lesions of the brain, or in experimental decapitation, hypertonicity of muscle does not appear suddenly, as might be expected on this hypothesis, but supervenes gradually and after a certain lapse of time; and although the original lesion which causes it may remain in statu quo and even decrease in energy, the muscular tonicity nevertheless continues slowly to increase. This condition, moreover, if developed, relaxes during sleep or chloroform narcosis when cerebral influences are temporarily in abeyance, a fact which is also opposed to this view. Again, if the cord is cut across, the sudden "let go" of energy ought to cause immediate tension of the muscles, which it does not at first (the contrary, in fact, takes place), and this only occurs after a considerable interval, and in a slowly progressive manner. How also on this hypothesis could the fact be explained that, when one side of the body has been affected by a lesion on the other side of the brain, generally before long the other side, though to a lesser extent, shows signs of augmented muscular tonicity? These, among other circumstances, would serve to indicate that, for the production of hypertonicity of muscle, some other agent is necessary than the mere release of physiological action by interruption of controlling influences from above. They would seem to show that, in the causation of the phenomena in question, some new and morbid active process in the cell elements is necessary, in addition to the mere freeing of restrained natural impulses.

A third theory, which more or less modified receives the support of Hughlings-Jackson, Bastian, and others, assumes that, when cerebral influence is suppressed, the uncontrolled action of the cerebellum (which is believed to be the excitor

of tonic muscular action) becomes predominant, and so produces the exaggeration of muscular tonicity. In health, it is supposed these two centres of innervation counterbalance one another. If the one is enfeebled, the other by retaining its natural vigour causes excess of action in that direction. The same arguments which can be urged against the last, seem to me to tell equally against this theory. Is there any positive evidence that the cerebellum plays any part in the production of muscular tonicity? Electrical stimulation of its substance certainly is not followed by any visible phenomena. How on this hypothesis can be explained the fact that, in complete division of the cord, when all connection with the cerebellum is severed, that hypertonicity of muscle subsequently becomes developed?

Such are the leading theories which have been advanced to explain the phenomenon of muscular hypertonicity, as well as some of the reasons to show, that no one of them by itself is adequate to account for its production. While it is not difficult thus to take a negative view of this complicated position, and to criticise existing doctrines, it is not so easy to successfully assume a constructive attitude, and to replace doubtful opinions by others more worthy of acceptance. One of the main impediments to arriving at sound conclusions, is the conflicting and contradictory statements regarding elementary physiological and pathological facts. But when the delicate and complex conditions which surround the nervous system are considered, it is not surprising that the results of experiment and disease should prove so difficult of exact definition. Reasons having been given for not admitting the Inhibition and Cerebellar theories, I am disposed to think that the irritation hypothesis of the French school, with certain modifications, offers the fewest objections to any yet advanced, and best explains all the facts so far as we yet know them. In the production of spastic paralysis, it is assumed by those holding this view, that the hyper-excitability of the ganglion cells is caused by the irritation of the contiguous sclerotic fibres of the lateral columns. Now it has been seen that muscular hypertonicity occurs too late after the central lesion to permit of its being explained by the Inhibition and Cerebellar theories;

on the other hand it takes place too soon to be satisfactorily accounted for by this one, namely irritation by secondary sclerotic processes. For, prior to demonstrable degenerative change in the lateral columns there are evidences of motor instability, as a few hours after an apoplexy exaggerated tendon phenomena gradually develop. It is therefore obvious that some change (as a result of removal of cerebral trophic influences) must exist in the motor tract before structural alteration can be appreciated. The nature of this change, whether vascular, molecular, or dynamic, we do not know; but that it is sufficient to produce objective functional symptoms in the shape of muscular hypertonicity is certain. A plausible explanation of this fact would be that vascular, or other nutritive modification of the pyramidal tract, was the almost immediate consequence of the disorganised trophic centres above, and that this change in the circulation or molecular stability led to irritable weakness of the adjacent grey matter of the cord. We know that cortical destruction subsequently leads to positive degenerative changes in the lateral columns; it is therefore not difficult to believe that, long before this is capable of demonstration, molecular or nutritional alterations may be produced by the same cause in the same parts, which if not capable of appreciation by our senses is sufficient to induce functional derangement of the regions with which they are connected. The same effects might supervene upon conditions other than those of actual gross cortical or pyramidal disease, as when the higher nervous elements without demonstrable anatomical change are functionally disordered. In such a case the actions of the parts below, which are dependent for their constancy on healthy influences from above, would be deranged. Thus from functional irritability or depression of the cerebral cortex there might arise instability of the pyramidal tracts and the motor apparatus connected with them, and, as a consequence, a train of typical hypertonic symptoms. Such excitants would be all the more potent in predisposed subjects whose spinal ganglion cells are already in an abnormally exalted condition, and whose muscles readily react to slight physical injuries. Under the same or similar conditions it is but a step further to realise, that psychical

influences might act as exciting agents for the production of like results. Thus, misdirected volition, emotion, or imagination acting as afferent stimuli might be the originators of abnormal motor phenomena, even when the nervous system itself was in other respects healthy. Again, even normal mental impulses might cause undue motorial demonstrations if the spinal centres were hyper-excitable, as is seen in strychnine poisoning, hysteria, &c. It is therefore obvious that if both these abnormal conditions existed together—that is, increased power of the higher centres to receive and reflect impressions, and augmented capacity of the lower centres to respond to psychical stimuli—the effects would be the more violent in degree and permanent in duration, and it serves to explain the severity and intractability of many functional disorders. This would also account for the fact that, under these circumstances, the physical motor symptoms are sometimes such as could not voluntarily be produced in health, as instanced by the phenomena of hypnotism and hysterical mimicry. It would also offer an explanation of the sudden onset, of the rapid and complete recovery which so often takes place in such cases.

Taking all these possibilities into consideration, I am inclined to think, that the hyper-excitability of the reflex spinal centres which is the fundamental element in the causation of hypertonicity of muscle, from whatever cause it arises, is an active rather than a passive process; that it is due to a new and abnormal irritation, the result of nutritional change, either directly and primarily affecting the cells themselves, or induced in them indirectly and secondarily by irritation of their afferent or efferent elements, or by interruption from the trophic centres with which they are associated. The whole progress of the condition seems to favour this view; its comparatively gradual onset, the fact of its being favoured rather than arrested by cerebral irritation and mental activity, its tendency to extend from one side of the body to the other when originally caused by an unilateral cerebral lesion, its aggravation under such circumstances as produce excitability, and its amelioration under opposite conditions, all point in the same direction. This irritative theory is substantially that of

the French school; but instead of speaking of degenerative processes as the cause, I would replace the term by that of nutritive alterations, an expression of wider scope, and better adapted to account for the varied forms of the disease.

In connection with these reflections it is a question of the greatest practical importance to know, whether repeated and permanent functional over-activity is capable of subsequently inducing demonstrable structural changes in the tissues affected. *A priori* reasoning would favour such an hypothesis, although there is not a sufficient series of recorded facts to make its demonstration certain.

To sum up the heads of this complicated enquiry: it is suggested that increased muscular tonicity, from whatever cause arising, is essentially due to abnormal hyper-excitability of the motor ganglion cells of the anterior cornua of the cord. The hyper-excitability may, for various reasons, be due to idiopathic alterations of the cells themselves, or may be induced therein by influences emanating from the brain, the cord, or the peripheral nerves. These influences are probably of an irritative nature, and may therefore be considered as a new morbid process. This irritation, when it exists in the pyramidal tract, may be excited there either directly by organic disease or functional excitement, or indirectly by changes in its nutrition as a result of severance from its trophic centres. These changes in nutrition may be either gross or dynamic. The gross changes usually follow gross disease of the cortical centres, while the dynamic changes may either follow their structural alterations or their functional disorder. Finally, this functional disorder of the motor tract may have its origin in the activity of psychical as well as of motor centres. These theoretical considerations concerning the pathology of hypertonicity of muscle are, like other hypotheses, open to criticism. I admit them to be somewhat involved, but this is scarcely to be avoided in attempting to account for the complicated circumstances connected with this group of symptoms.

If such doctrinal speculations on the subject we are discussing are uncertain, the practical difficulties met with in actual clinical observation are none the less perplexing. As their features are now well recognised, I have passed over those

forms of muscular hypertonicity associated on the one hand with secondary changes from gross disease of the nerve centres, and on the other with obvious functional disorder. I have limited the practical question to the uncomplicated form of the affection described in the first part of this paper. Now, I would ask, what is the anatomical substratum of this condition? Is it a so-called functional disorder—that is, one unaccompanied with discoverable tissue change—or is it the result of gross organic disease; and how is this to be determined during the life of the patient? Where, and in what manner, does the affection originate? Is it a primary disease of the brain, cord, or nerves? What are its causes, by what criteria are we to be guided in framing a prognosis, and what is the suitable treatment? Although I have seen many such cases and have followed the progress of some of them for years, I confess I am unable to give a satisfactory reply to the above enquiries.

The classical morbid type which this condition most nearly resembles is the primary spastic or spasmodic paralysis of Erb and Charcot, which is asserted to be caused by primary sclerosis of the lateral columns of the cord. There are, however, certain considerations which render it impossible for the two to be considered as identical. The essential elements of spasm and rigidity of muscle in the one are, as we have seen, not necessary features of the other. It is of such frequent occurrence, it so often ends in recovery, and its course and termination are sometimes so rapid, and influenced by such circumstances, as to render its explanation by permanent organic degeneration of the pyramidal tract impossible. That a close resemblance exists between these two conditions cannot be doubted, and it is probable that the essential phenomena of both have the same pathological significance. It may even be asserted that the one is merely an initial form of the other; but this is just the point which yet remains to be proved, and which constitutes the real question at issue. We find, however, that the one may exist independently of the other, and may therefore be said to represent a separate clinical state. Again, it might be maintained that the condition is an incipient stage of some gross form of disease, such as disseminated sclerosis, or the secondary results of primary lesion elsewhere. It can only

be replied that, in typical cases, no evidence can be traced of any such connection.

Finally, to place such cases under the category of Hysterical or Functional, is simply to shelve the difficulty, and brings us no nearer the true explanation. For convenience of recognition the clinical group of symptoms might be named "*Hypertonic paresis or paralysis.*" It is not pretended that this term is a perfect one, or that it represents a true disease. It professes only to be a convenient symbol expressive of a group of symptoms, frequently met with in practice, the causes of which are probably various, and of the nature of which we are ignorant.

In conclusion, I trust that this communication will serve the purpose for which it was undertaken, namely, by indicating certain obscure problems of practical and scientific interest, to elicit the opinions and experience of the members of the Neurological Society. As a guide to the debate, I beg to suggest the following propositions for special consideration, which provide a wide and varied field for discussion on the subject, from every point of view.

I. *With regard to the clinical condition of so-called Hypertonic Paralysis.*

1st. Is the existence of the clinical type admitted? If so, what is its anatomical substratum? Where, and in what manner, does the affection originate? Is the primary disturbance in the brain, cord, or nerves? Is it due to gross organic lesion, or the result of so-called functional disorder?

2nd. What are its causes?

3rd. What is its prognosis? By what criteria are we to foretell in individual instances whether the affection will be temporary and comparatively innocuous, or whether it is permanent and incurable?

II. *With regard to the Physiological and Pathological problems in connection with muscular hypertonicity.*

1st. What hypothesis best accounts for hypertonicity of muscle?

2nd. Admitting that visible organic degeneration of the lateral columns is frequently associated with the production of hypertonicity of muscle, is this an essential element for its

production? May a like condition result from undemonstrable nutritive modifications in the same regions or in the psychical or motor centres.

3rd. May a primarily so-called functional disorder, consisting of definite symptoms without appreciable tissue change in the nervous system, eventually lead to demonstrable structural degeneration?

DISCUSSION.

Dr. Hughlings-Jackson.

I feel grateful to Dr. Hughes Bennett for bringing together so many varied facts in so methodical a way. I wish first to remark on a common use of the term functional—not on that use of it by Dr. Bennett only, but on its use by most physicians. It is sometimes used as if there could be nervous symptoms, morbid manifestations, without abnormal changes in the nervous system. As Dr. Allchin has ably and vigorously contended in the second volume of the 'Westminster Hospital Reports,' this use, or rather misuse, of the term is to be deprecated. Some cases called functional are, I think, cases of deliberate imposture: for example, cases of boys or adults who do not talk at all, but eat and swallow well, who can write, and who are suddenly or rapidly "cured" by impotent treatment. There are no "abnormal changes" in these cases, which I believe, as I said, in effect, to be neither of pathological origin nor hysterical, but to be nothing at all. I believe, too, that many cases *called* hysterical are cases of mere pretence. The word functional is sometimes otherwise used; it is used for cases in which morbid changes in the nervous system, or some part of it, are supposed to be very slight. If a patient has hemiplegia for a few hours only, his case may be called functional. For my part, I should have no doubt whatever that there was a slight lesion, whether it might be discoverable (if we had subsequently a post-mortem examination) or not.

I have long urged that the term functional should be used as the adjective of the word function. (I remark, parenthetically, that such expressions as that "consciousness is a *function* of the brain," or of any part of it, are illegitimate. Consciousness *attends* functioning of the brain, or of some parts of it.) Function is a physiological

term: it deals with the "storing-up" of nutritive materials having potential energy, with nervous discharges (or liberations of energy by nerve-cells); it has to do with the rates of those liberations, with the resistances encountered, and with the different degrees of those resistances. Abnormalities of function are of two kinds, minus and plus. There are degrees from defect to loss of function. After an epileptiform seizure beginning in one foot, there may be paralysis of the leg; in this case there is loss or defect of function of some fibres of the pyramidal tract—exhaustion of them consequent on the prior excessive discharge beginning in some part of the mid-cortex. In cases of hemiplegia from breaking-up of the internal capsule by clot or softening, there is destruction of fibres and also, of course, loss of function. There is an opposite or plus kind of functional change—a departure from normal function by excess; this also implies alteration of nervous elements. For example, there is in epileptiform seizures what I call a "discharging lesion," or "physiological fulminate," or, to use Horsley's term, an "epileptogenous focus"; in these cases, cells of a part of the patient's mid-cortex have, by some pathological process, attained unduly high instability, issuing in occasional sudden, excessive, &c., discharges. We ought, I think, to endeavour to distinguish most carefully abnormal functional—that is, abnormal physiological—changes from the pathological processes leading to them. A "discharging lesion" is an hyper-*physiological* condition; the pathological process, disorder of nutrition, which produces it, is a thing for separate consideration. This I have long urged, and I now urge it again, because I have been said to put forward the "theory of discharges" as the *pathology* of epilepsy; the real fact is, that I have urged that the functional, or abnormal physiological, change is *produced by* some pathological process. When a cortical tumour is said to be the "cause" of epileptiform seizures, we must bear in mind that the direct cause of the fits is really the hyper-physiological condition which I call a "discharging lesion"; this is produced by the tumour in its general character as a "foreign body"; probably the tumour induces a local encephalitis whereby some cells are destroyed and others are rendered highly over-unstable. In the case of hemiplegia, when the internal capsule or part of it is destroyed, the distinction between loss of function and pathological process may seem to be, but is not really, frivolous. Putting the case otherwise: the hemiplegia spoken of is not, properly speaking, a symptom of clot, softening or tumour, but is a symptom of destruction of fibres of the internal capsule *however produced;* hemiplegia may be a temporary result of mere exhaustion of those fibres, temporary loss of their function by a

prior excessive discharge (*vide infra*). In the same way an epileptiform seizure is not a symptom of cortical tumour, or of changes in the cortex, produced by embolism, &c., but of a "discharging lesion," *however produced*.

For the sake of completeness I remark, that a knowledge of the anatomy of cases of nervous disease is a knowledge of the parts of the body which the nervous arrangements in abnormal function, minus or plus, represent, and of the ways in which they represent those parts. Anatomy should not be confounded with morphology. Before the researches of Hitzig and Ferrier the anatomy of the convolutions of the Rolandic region was, according to the opinion of nearly all physicians and physiologists, unknown; we had only morphological knowledge of them. The clinical problem in any case of nervous disease is one in Anatomy, in Physiology, and in Pathology. I do not mean simply, what is obvious, that we require anatomical and physiological knowledge in order to properly analyse cases of patients. I mean, to take an example, that to discover the seat of the lesion productive of fits beginning in the right thumb is itself an anatomical affair; that questions about the spasm, as to its rapidity of onset and the speed of its spreading, being indicative of central functional abnormality (excess of function), are questions in physiology—abnormal physiology, if that term be preferred—and that facts or hypotheses bearing on the abnormal nutritive process by which the functional lesion is produced are alone questions in pathology. I hope the foregoing will not be taken as too much of a digression; it is desirable that the term functional should be used with exactness, and that functional (physiological) changes should be distinguished from the pathological changes producing them. I now come close to the subject of Dr. Bennett's paper, taking, however, for the present but a single symptom—increase of the knee-jerks.

One opinion is that the increase of knee-jerks, say in an ordinary case of hemiplegia, is the direct result of some kind of pathological process; thus it is supposed by very many physicians that the process of "descending wasting" which destroys fibres in the pyramidal tract, when it reaches the anterior horns produces in their cells the exactly opposite physiological condition, that of exaltation of function. Against this view is the fact that, in some cases of hemiplegia, there is an even, slight wasting of the muscles of the paralysed limbs, evidence that the "descending process" can lead to atrophy and consequent loss of function of a few cells of the anterior horns. This renders it unlikely that the same process produces also the opposite effect; yet it may be reasonably contended that small cells are destroyed when the large ones are

rendered more excitable. I have put forward the speculation, that the anterior horns in the cases mentioned are in exalted activity from loss of control—that this exaltation is not caused by any direct pathological process.

The preceding remarks on *plus* functional changes I illustrated by "discharging lesions" directly produced by pathological changes. But I now mention that there is another kind of *plus* functional change—a much slighter one—which is not produced by direct pathological processes. Increased rate of cardiac action after section of the vagus is a commonplace example; but the same principle, increased activity from loss of control, otherwise of inhibition, is displayed in very many different cases of nervous disease, as from fibrillar contractions in some cases of progressive muscular atrophy to post-epileptic mania. The slight hyper-physiological states from loss of control ought to be very carefully distinguished from those vastly greater hyper-physiological states called "discharging lesions." Increase of the knee-jerks in cases of hemiplegia is, for me, simply an hyper-physiological condition; although this, of course, involves (although it is not caused by, or is not primarily the result of) increased nutritive changes. Being "cut off" from the higher (cerebral) centres, the anterior horns in question are "let go": they gain autonomy. The pathological process is that which cuts their connection with the middle motor centres ("motor region"); is that which destroys fibres of the internal capsule. I have suggested, too, another factor; that by the taking off of cerebral influence from the anterior horns, the cerebellar influence on them is no longer antagonised. I shall say nothing for the present of this hypothesis.

The objections to the hypothesis stated are obvious. Although increase of the knee-jerk is sometimes found directly after the lesion of the internal capsule, it mostly, so to speak, "waits for" the "descending wasting." We have, however, to study more widely. Besides cases of hemiplegia and any other cases in which there is lateral sclerosis, we have to note conditions of the knee-jerk after epileptiform seizures. I have published[1] the case of a patient who had exaggerated knee-jerks and foot-clonus of the left leg, which limb was temporarily paralysed after an epileptiform seizure beginning in the left foot, and which affected the left leg and but little more.[2] In this case it is perfectly certain that during the paroxysm there was an excessive functional condition from the part of the mid-cortex discharging to the particular

[1] "Med. Times and Gazette," Feb. 12th, 1881.

[2] Are there altered tendon reactions after hysterical or so-called "functional," fits?

muscles convulsed; it seems reasonable to infer that the local paralysis after the fit signified exhaustion of corresponding fibres of the pyramidal tract—a negative, or, as we said, a *minus*, functional lesion, equivalent *for the time being* to the destruction of the fibres. If so, a state of things was rapidly produced which is slowly produced by "descending wasting" in ordinary cases of hemiplegia. Both kinds of cases have to be taken into account by any hypothesis concerning increase of the jerks. But how are the two to be reconciled on the hypothesis of "loss of control"? Gowers has suggested, that close upon the anterior horns are small inhibitory centres, and that these are destroyed by the "descending wasting," whilst the anterior horns themselves ("muscle centres") are uninvaded by the pathological process. We may explain the increase of the knee-jerks in cases of post-epileptiform paralysis of the leg by the supposition, that the small inhibitory centres are alone exhausted, the muscle centres not being exhausted. It is a question of the severity of the prior fit. For after some epileptic fits[1] the knee-jerks are lost for a very short time. Beevor, who has carefully noted the condition of the knee-jerks after epileptic fits, in many cases has found exaltation, but in some loss, of the knee-jerks. In cases of post-epileptic loss of the knee-jerks, the presumption is that there is deeper exhaustion, not only of Gowers' small inhibitory centres, but of the "muscle centres" also.

I by no means think that the hypothesis, so far as it is stated provisionally, accounts for all the facts. I have reported a case of loss of both knee-jerks in a case of hemiplegia, in which case Dr. James Anderson discovered no coarse or microscopical lesion in the lumbar enlargement. Dr. Stephen Mackenzie has recorded a case of hemiplegia with loss of both knee-jerks, and has stated an interesting hypothesis bearing on the interpretation of such cases. I now approach the general subject under discussion from another standpoint, and shall consider the possibility of cerebellar influence being a factor in causing the super-positive effects, increase of tendon-reactions and rigidity.

Rhythmical tremor and rigidity are degrees of one thing. I have submitted the hypothesis, that in paralysis agitans there is wasting of cells of the middle motor centres (so-called "motor region" of the cortex), in order from smallest towards largest cells. Such a process, a negative one, can cause only the negative symptom paralysis. But being at the same time a loss of control over anterior horns, there is, when but a few cells are wasted, slight over-activity of anterior horns; there is rhythmical move-

[1] Westphal, Gowers and Beevor.

ment (spinal chorea). When more cells of the middle motor centres are destroyed there is more paralysis; the anterior horns being still less controlled, there results ultimately rigidity of the parts previously tremulous. It has been said that the head and neck escape tremor; this is perfectly true in a superficial sense. But these and other parts escape tremor as the proverbial fish escaped the frying-pan: they escape tremor by passing straightway into what is a higher degree of tremor, rigidity. I have suggested that the cerebellum is partly concerned in producing the tremor and rigidity of paralysis agitans, and also the rigidity in ordinary hemiplegia. On taking off of inhibition from the anterior horns the cerebellar influence on those horns is supposed to be no longer antagonised, and by this influence the tremor and rigidity are partly accounted for. I submit that the cerebrum represents all parts of the body, and that the cerebellum also represents all parts of the body. But the two representations are in inverse order; putting it roughly and neglecting some parts, the cerebral order is, arm, leg, trunk. This is admitted when we say that the "brain is the organ of volition," for the order stated *is* the order of parts as they are most often used voluntarily. The cerebellar order of representation is the opposite—trunk, leg, arm. This is admitted by those who say that the cerebellum is the organ "co-ordinating locomotor movements," for that expression only means an organ where muscles of all parts of the body are represented in certain complex ways. The order of parts getting into play in locomotion, from walking to swift running, is, trunk, leg, arm. Paralysis agitans is certainly a morbid motor affection in cerebral order; an advanced case *is* double hemiplegia with rigidity. And we note that those parts suffer tremor before rigidity which are most under cerebral and least under cerebellar influence (fingers, hand, arm); and that those parts (neck and back), which are least under cerebral and most under cerebellar influence, pass into rigidity without a prior stage of tremor. That both the cerebrum and cerebellum are engaged in any operation will, I suppose, not be doubted. In manipulating, the cerebrum will be only *chiefly* engaged; in walking, the cerebellum will be only *chiefly* engaged. There is co-operation of amicable antagonism between two large divisions of the nervous system representing the body inversely. I wish now to speak of Cerebral and Cerebellar Inverses from disease. Hemiplegia *is* the inverse of cerebellar paralysis; in the former the order is arm, leg, trunk, in the latter, trunk, leg, arm. The attitude (emprosthotonic) of an advanced stage of paralysis agitans *is* the inverse of the attitude, opisthotonic, in the paralysis and rigidity of some cases

of tumour of the middle lobe of the cerebellum. The commonest variety of epileptiform seizures, one beginning in the hand, is the inverse of certain tetanus-like seizures in some cases of tumour of the middle lobe of the cerebellum.[1] I think, too, that we have inverses on the lowest level. The pons, medulla oblongata, and spinal cord (together one evolutionary system) are cerebro-cerebellar; some lowest centres for arms on this level will be most under cerebral influence, and others for trunk, most under cerebellar influence. There is a progressive muscular atrophy (I have called it bilateral) which affects the trunk and the upper parts of the limbs most, never, in my experience, reaching the hands. It is bilateral in fact, corresponding muscles on both sides being affected at the same time; it is, so to speak, bilateral "in intention," the muscles which act together, or in necessary alternation, being those suffering first and most. This kind of muscular atrophy is, however, said to be, or to be often myopathic. At any rate, it is roughly the complementary inverse of the ordinary Duchenne-Aran type of progressive muscular atrophy, which mostly begins in one hand, begins unilaterally, and which affects first and most parts which are unilateral in most of their activities.

I do not mean that the hypothesis of loss of control and of unantagonised cerebellar influence has not many facts against it. I bear well in mind that, on section of the cord in animals, there is increased reflex action of parts below the section. But I hope the suggestions I have made are worth taking into account. I admit great difficulties, and come to no conclusion, or only to the provisional conclusion which is called hypothesis. I wish to draw particular attention to some cases recorded by Bastian. He has found that in some cases with incomplete transverse lesions of the cord there has been exaltation of the knee-jerks, and that in the same cases upon the lesions becoming completely transverse there was loss of the knee-jerks. Again in some cases of *complete* transverse lesion of the lower cervical cord from fracture dislocation there is loss of the knee-jerks. "Shock" cannot be invoked to explain this loss, in cases where the patient has lived several months after the accident, without a trace of jerk being obtainable.

Dr. Buzzard.

There can be no doubt, I think, about the existence of the clinical type which Dr. Bennett has described with so much graphic power in his highly interesting and instructive paper. Cases of the

[1] I have suggested that ordinary surgical tetanus is owing to morbid changes of the cerebellum; probably of the pons medulla and cord too

sort which he has pictured are indeed very numerous. It is doubtless true that the majority of them have been roughly classed—many rightly, some wrongly—along with others of a somewhat different kind, as examples of functional or hysterical paraplegia, and I have long been disposed to think that some of them would be best explained by the supposition, that an incipient sclerosis of the lateral columns stopped short and was recovered from in an early stage. For my own part, I would still prefer to wait till something more is learned of their pathology before applying to them the proposed title "hypertonic paresis or paralysis," a designation which, whilst in my opinion it does not quite accurately describe the condition, might possibly cause these cases to be confounded with Thomsen's disease, with which I need scarcely say they have nothing in common.

Certain general propositions of a physiological nature are formulated by Dr. Bennett, and these we are invited to accept before entering upon the discussion of the matter more particularly. Unfortunately for me, I find myself unable to accept even the first of these propositions. My difficulty is this.

"Healthy muscle," Dr. Bennett remarks in his first proposition, "contracts when its fibres are subjected to sudden stretching (extensile stimulus). This may be induced by percussion on its substance, and still more effectively by a blow on its tendon, the last constituting the so-called tendon-phenomenon. The mechanical condition necessary for the production of the response following percussion is sufficient passive tension of the muscular fibre.

"This property," he goes on to say in the second proposition, "is intimately associated with and obeys the same laws as what has been termed reflex muscular tonicity, or that state of slight constant tension which is the characteristic of healthy living muscle. Both are abolished when there is interruption at any portion of the reflex arc of which muscle is the terminus."

Now if these propositions were correct it would necessarily follow that, whenever direct percussion upon a muscle produced contraction, the tendon-phenomenon belonging to that muscle would be preserved, because the sudden stretching of a muscle by percussion of its substance is still more effectively brought about by a blow on its tendon. Unless I have failed as to meaning of the words employed, this is what the author intends to affirm. But, as a matter of fact, what is the case? Whilst there is no doubt that, as a general rule, the association of events described is observed—that when you percuss the plump of the vastus internus muscle and find a good contraction you expect also to find the knee-phenomenon produced by tapping the ligamentum patella—yet exceptions to

this rule of the most striking character occur, so striking and significant as, in my opinion, to throw strong doubt upon the correctness of the opinion, that the tendon-phenomenon and the muscular-percussion phenomenon are of the same nature. In tabes, for example, where the tendon-phenomenon is entirely abolished, percussion on the vastus internus itself invariably produces contraction of the muscle, which is very often, as I pointed out many years ago, more strongly marked than in health. If the muscular "tonus" is sufficient to allow of a vivid response of the muscle to the sudden stretching of its fibres produced by percussion of its substance, how is it that it does not suffice to permit of contraction when the sudden stretching is produced by a means which is said to be more effective than percussion?

On the other hand, in many cases of muscular atrophy associated with bilateral sclerosis, whilst the tendon-phenomenon is enormously exalted, response to percussion of the muscular substance itself may be almost nil.

Again, I showed before this Society in January last a case of the so-called Thomsen's disease—an affection characterised more than other by an exaggerated tendency of the muscle to strong and enduring contraction in response to any stimulus. The patellar-tendon phenomenon was absent, instead of being, as it should have been (were the author's proposition correct), greatly heightened.

It may reasonably be urged, however, that the proposition is intended only to apply to healthy muscle, and that I have no right to criticise it when the question of the muscle of an unhealthy person is under consideration. Now we have certain tests by which healthfulness of muscular structure may be presumably ascertained, and yet we find that, even in a case where these tests have been satisfactorily applied, the views formulated in the proposition are distinctly contradicted. Here is an example: In a case of tabes the knee-phenomenon was absent in one leg, but preserved in the other. The voluntary power, as ascertained by resistance to passive movements, as well as by the patient's own description of the facility of active motion, appeared to be equally good in the two limbs. A blow upon the vastus internus of either limb caused an equal and, to the eye, precisely similar contraction in each, and so did the application of induced currents, whilst on either side the response to the closure of a voltaic current was precisely similar, and of absolutely normal character. Is it likely, I would ask, that the knee-phenomenon is a mere question of contraction following the sudden stretching of a muscle which is in a state of the requisite muscular tonicity when we find, as in

this case, its absence in one limb, and its presence in the other, associated with a state of muscular fibre which all our tests—voluntary action, percussion, state of nutrition, electrical reaction—show to be in a similar and normal state in each limb?

In cases of lateral amyotrophic sclerosis we may not unfrequently find the wasted and degenerated muscles hanging flabbily from the thighs, and associated with this a highly exaggerated knee-phenomenon. Can this be properly described as a condition of muscular hypertonicity?

Such are some of the difficulties from the clinical side which interfere with my acceptance of the view, that the so-called tendon-phenomenon is but the contraction of a muscle submitted to sudden stretching, its fibres being in the requisite state of tonicity. As I do not admit the soundness of the propositions to which I have taken exception, I cannot of course assume with the author that increased tendon-phenomenon is due to hypertonicity, and that the essential factor for its production is an exaggerated excitability of the ganglion cells of the cord. It is unnecessary, therefore, for me to discuss Dr. Bennett's view, as to the mode in which this assumed abnormal excitability is brought about.

Let me turn, for a very short time, to the clinical type of disease which the author has described—cases in which more or less impairment of voluntary power in the lower extremities is associated with exaggerated tendon-phenomena. The general correctness of his descriptions of such cases every one of us, I suppose, will be ready to admit. There is one point, however, in which his experience apparently differs from mine. He speaks of the superficial reflexes being "usually normal, if anything, slightly increased." According to my observation the plantar reflex is either altogether absent or but very feeble in a large proportion of these cases. So striking has this appeared to me, that I have long been in the habit of teaching in the hospital that the association of exaggerated knee-phenomenon and ankle-clonus with absent or very weak plantar reflex (cutaneous sensibility not being materially affected), should at once suggest the probability that the case so marked is one of functional character. Need I say that this is not of itself conclusive, as a localised myelitis might produce a similar combination? But were the latter lesion present other symptoms would be found, which I need not here dwell upon, and which would leave the matter in no doubt. I submit that the association described should always suggest the existence of functional paralysis.

Returning here for a moment to the author's views, I would urge that the absence or enfeeblement of plantar reflex is a

symptom which can hardly be said to point to that exaggerated state of excitability of the ganglion cells of the cord, which is assumed by Dr. Bennett to be the essential factor of the increased tendon-phenomenon observed in the affection which we are discussing.

I freely admit, whilst insisting upon points which appear to me to militate strongly against the author's views, that the absence of plantar-reflex alongside of exaggerated knee-phenomenon and ankle-clonus so frequently presented in these cases, is anomalous from whatever point of view the tendon-phenomenon is regarded. And there is another circumstance of paradoxical character to which I should like to allude whilst upon the subject.

In cases of spastic paraplegia from bilateral sclerosis the exaggeration of tendon-phenomenon is usually confined to the limbs which are the seat of muscular weakness. As a general rule, not however without its exceptions, where the lower limbs alone shew loss of power, whilst the knee-phenomenon is lively in the extreme, and there is most characteristic ankle-clonus, there is an absence of supinator longus reflex at the wrist, or if this be present it is not strongly expressed. In cases of the kind, however, which we are considering, and which are probably free from, at all events, gross organic lesion, we continually find that not only are the lower extremities (which are affected with marked muscular weakness) the seat of exaggerated tendon-phenomena, but the upper extremities also, about which no complaint has been made, and which the patient, on enquiry, may allege to be perfectly normal, display an increase of tendon-phenomenon quite on a par with that which marks the lower extremities. In such a case it has always appeared to me legitimate to "discount," as it were, the clinical value of the exaggerated reflex displayed by the lower extremities. I have very frequently, indeed, acted upon this principle, and cannot remember an instance in which I had cause to regret the conclusion to which it led me, that the loss of power was not due to organic disease of the cord.

In some few of these cases of loss of power with exaggerated tendon-phenomena, I have found a condition of partial optic atrophy of simple character accompanied by some amblyopia. When the question of adopting the Weir-Mitchell treatment in such examples has been considered, the existence of the optic atrophy has been with me a reason for deciding against the employment of this treatment, owing to the presumption of some organic change being present. I am not quite sure that the inference is justified. It occurs to me as possible that there may be in certain of these cases a phase of optic atrophy as much

amenable to treatment as that which owes its origin to excess in tobacco. At present, however, I have seen no evidence of this. Indeed, in one instance of the kind in which, influenced by the appearance of the optic papilla, I had, though with considerable hesitation, advised against the Weir-Mitchell treatment, the patient eventually underwent the process with a certain amount of benefit, which, however, as I am informed, was not permanent.

Dr. Ferrier.

On this question of exaggerated tendon reaction and muscular hypertonicity, I am in all essential particulars in accord with the views and arguments so concisely and lucidly set forth by Dr. Bennett. The reflex character of the tendon reaction is, I imagine, now generally abandoned. The most conclusive disproof of this theory is furnished by exact measurement of the time that elapses between the percussion of the tendon and the muscular contraction. It has been shown by the researches of Waller, and other observers, that the muscular response is more rapid than the most rapid reflex reaction. Waller has also demonstrated that the time is the same for all muscles irrespective of the length of nerve traversed, and further, that the time is precisely the same as that of muscular response to direct mechanical exaltation.

That the hypertonicity or contracture of muscle is the result of some nutritional change in the anterior horns, directly or indirectly induced in the ways enumerated by Dr. Bennett, but not necessarily of a structural character, *i.e.* without demonstrable degeneration, is, I think, beyond doubt. That increased tendon reaction and ankle clonus are not necessarily indicative of structural degeneration, is to my mind conclusively demonstrated by the results of clinical investigation, and I should regard the opposite view as one likely to lead to serious errors in diagnosis and prognosis. If it be asserted that the fact of recovery from such a condition does not indicate the absence of structural degeneration, then it must be a form of structural degeneration quite unlike all others with which we are acquainted.

Dr. Bennett has adduced cogent arguments against the explanation of increased muscular tone by the cutting off of some hypothetical inhibitory centres. I am myself greatly opposed to the multiplication of 'centres' over and above those concerned with sensation and motion, general and specific, and to the assumption in particular of the existence of centres whose specific function is to inhibit or restrain the action of others below them. These

inhibitory centres would, according to the views of some writers, require to be almost independent intelligences, and would involve a system as complicated as the cycles and epicycles of the Ptolemaic astronomy. That nerve centres detached from their connections are more mobile and excitable than when they form parts of a complex organisation, is more probably a pure question of physiological physics than anything else. And it is doubtful whether there are better grounds for assuming that higher centres inhibit lower centres, than that the lower exercise an inhibitory influence on those above them.

Be this as it may, the hypertonicity of muscle cannot be explained by the cessation of a supposed inhibitory influence excited from above, and this is shown more especially by the fact, that the contracture supervenes gradually and not suddenly when the supposed controlling centre has been removed. I have elsewhere ('Localisation of Cerebral Disease') urged similar objections to those used by Dr. Bennett against the cerebellar theory of late rigidity advocated by my esteemed colleague Hughlings-Jackson. Dr. Bastian, however, is of opinion that this theory derives strong support from the facts of a case which he records in his recent work ('Paralysis, Cerebral, Bulbar and Spinal,' p. 221). This was a case of partial paraplegia with exaggerated tendon reaction in the legs, in which the hypertonicity disappeared with the setting in of complete transverse myelitis in the dorsal region. There is, however, no doubt that cases of transverse myelitis occur in which, notwithstanding the complete severance of the cord, exaggerated tendon-reactions and contracture are manifested.

Inasmuch as this is so, it is more probable that in the case related by Dr. Bastian, coincidently with the complete softening of the cord in the dorsal region, there occurred some morbid process in the anterior horns of the lumbar region. Such a morbid condition sufficient to abolish the tendon-reaction may exist without obvious anatomical change, as is exemplified in Landry's paralysis. The fact that hypertonicity and contracture may occur where there is complete severance of the cord proves that these conditions are not dependent either on cerebral or cerebellar influence for their manifestation.

I must, however, admit that I have seen cases of disease in which, from their symptomatology, the lesions were probably in the region of the pons-medulla, where the tendon-reaction has been entirely absent. And I have been able as yet to satisfy myself as to the exact pathology of such cases.

It is not, however, the fact, that lesions in this region always cause absence of tendon-reaction. And it is certain also that

increased tendon-reactions may be exhibited when the cerebellum is extensively destroyed. Facts like these render it probable that the absence of the tendon-reaction in certain cases of disease of the pons-medulla may be dependent on complications and not on the locality of the lesion as such.

On the clinical part of Dr. Bennett's paper, I would remark that I am familiar with the class of cases which he has brought under our notice, and I must confess that I am frequently in great doubt as to their exact nature and prognosis. I bring before you an instance of the kind :—that of a young woman who for the last two years has been suffering from symptoms of paraplegic contracture, spastic gait, and knee and ankle clonus of characteristic and extremely marked character [Dr. Ferrier here demonstrated the clinical symptoms of the patient in question]. In a large majority of the cases—and it is so in the patient before you—there is certainly an emotional instability of greater or less degree, and when such is clearly manifest, I am inclined to look upon them as cases merely of neurasthenia—meaning by that purely functional disorder. In such cases too, there is usually a great variability and want of continuity in the symptoms. The clonus and rigidity are altogether out of proportion to the duration of the disease, when one compares it with the progress of an undoubted case of lateral sclerosis. In all cases, of paraplegic contracture, I attach great importance to the existence or otherwise of relative weakness or paralysis among the various muscular groups. Where there is distinct paresis or paralysis of the anterior tibial group of muscles, though the limb otherwise seems fairly serviceable, I should regard the case as probably dependent on organic degeneration.

Whether the nutritional or molecular alteration of the anterior horns which we assume to be the basis of the manifestations of contracture and increased tendon-reactions may, if long-continued, result in demonstrable structural degeneration, is a question difficult to answer. For it would always be possible to argue, that the degeneration had been the cause of the symptoms from their commencement. But I am myself inclined to believe, that structural degeneration may in some cases be the result of long-continued nutritional excitability of the anterior horns, and that therefore 'functional' cases may occasionally pass into incurable structural degeneration.

Dr. Donkin.

I fully recognise the clinical existence of the class of cases brought forward by Dr. Hughes Bennett, and have been in the habit of pigeon-holing them provisionally, though I admit not without inaccuracy, with so-called 'spastic' paralysis. I have been aware, nevertheless, that many cases go on for an indefinite time, and that others recover, partially or completely, without showing any rigidity or unprovoked spasm. Dr. Bennett has, I think, made an addition to the clinical picture of these cases, as generally drawn, by calling attention to the frequent involvement of the arms and of several muscles of the body other than those of the legs. In most instances of this affection which have come under my notice, the legs have been mainly, or alone, affected; though I have seen several cases such as Dr. Bennett alludes to. I have but little doubt that some of these cases run on to the state of rigidity and spasm, and are then called spastic paralysis with more accuracy, or by some, I think rashly "lateral sclerosis." I have seen such progression, the symptoms being at first mere weakness with increased "reflexes," then startings of the limbs in addition—even when the patient is lying quietly in bed—and ultimately marked rigidity. Other cases, of apparently exactly similar nature at the outset, I have seen improve greatly after rest, and sometimes only after prolonged rest, in bed. The cases which recover are by no means all of them connected with any symptoms which may fairly be called "hysterical." The disorder seems to me to be quite as common in men as in women; and certainly a remarkably large number of the cases involving the legs occur in individuals who have used their limbs, either in standing or forced athletic exertion, to an inordinate extent. I have seen a few cases in children, one especially in a girl ten years old, which must certainly be regarded as belonging to the hysterical category. One leg only was affected when she came under my care, though both had been previously involved. There was a considerable degree of what Dr. Bennett terms hypertonicity; and at last there was great rigidity and apparent shortening of the leg owing to a tilting of the pelvis. This case was permanently, or at least completely cured after the administration of chloroform, much against the patient's will, for the purpose of examination of the rigid limb.

I have called attention to this class of case with instances of improvement with rest, in the October number of 'Brain' for 1885; and also in an article in 'Brain' in January, 1883. Since

writing these papers I have noticed in some cases the sacral pain mentioned by Dr. Gee.

From what I have seen and heard and read of the subject, I do not think that the disorders under discussion can be explained by any recognized lesion in morbid anatomy. I believe many of them are, from their probable ætiological connection with over-use of the affected limbs, incapable of such explanation; nor can I regard the formula of "excitability" or "irritation of the anterior cornua" of the cord as really anything else than somewhat pedantic tautology, or a mere statement in other words of the symptoms to be explained. Doubtless some of these cases, and notably some which recover, are attended by other symptoms obviously "hysterical," and are probably of this nature, whatever it may be. These should not at least puzzle us any more than the well-known case of hysterical paralysis without hypertonicity or spasm, difficult of explanation though these latter, as well as the former, may be. It would seem improbable that all cases of this disorder can be referred to the same category: they probably have different causes. Certain of these appear to be fairly explicable by the theory of loss of control and a disproportionate cerebellar efflux. But at present, and probably for a long time to come, the question of the pathology of these cases will be merely speculative. Morbid anatomy is unlikely to reveal anything explanatory, not only from the clinical nature of the symptoms, but also because of the few opportunities of post-mortem examination which are offered to those who might be sanguine enough to expect any discovery therefrom. The structural changes which underlie or accompany the functional disorder, in whatever part of the nervous system they may occur, will probably be more fittingly spoken of as molecular than in terms of our present or even our future knowledge of the pathology of cells, fibres, or neuroglia.

The most important practical point about these cases seems to me, at present, to be that of their clinical ætiology. The prognosis and treatment of each case depends in a great measure as to how far it may be referred to a *primary* and undiscovered, or undiscoverable, morbid change of nerve-centres, or how far to exciting causes arising directly from the external world, or from the action of the environment on the possibly predisposed organism, as the modern neurologist would express it. I think, as I have already said, that this latter hypothesis has some important facts in its support, as at least partially covering these cases; and that it throws some light on the proper management of some instances of this class of disorder, which has been so clearly and now for the first time minutely defined by Dr. Hughes Bennett.

DR. HALE WHITE

referred to the fact, that Dr. Fagge had been accustomed to teach that many cases of spastic paraplegia could be cured by the extract of Calabar bean, in doses of one-sixth of a grain gradually increased up to one grain thrice daily. Many of Dr. Fagge's cases were more severe than those shown this evening, and perhaps some were not identical with, but only allied to them. The last case mentioned by him in his "Principles and Practice of Medicine" was strikingly like some of those now exhibited. Dr. Hale White considered these cases to be functional, for they were often sudden both in their onset and cure, their severity varied from time to time, and they were often associated with other functional symptoms, as hysteria. He did not at all agree with those who said that to call them functional was begging the question. It is a distinct advance if, by reasons such as those just given, it can be proved that the disease can have no morbid anatomy visible even to the eye, aided by a microscope, which is what is usually understood by the term functional. They were separable from organic spastic paraplegia due to primary sclerosis of the lateral columns, because the latter was always gradual in its onset, did not particularly affect neurotic subjects, had no remissions, only spread up to the brachial region after many years, and was sometimes accompanied by incontinence of urine and optic atrophy.

DR. BENNETT'S REPLY.

The very brief time at my disposal allows me to do little more than thank the members of the Society for the indulgent manner in which they have received my opening address. All the speakers who have followed have provided much valuable matter for thoughtful study and reflection; and did opportunity permit, I might, perhaps, with profit emphasize and develop their views. The whole subject, however, is too extensive and complicated to warrant my occupying the time of the Society with further details, and as fortunately the leading principles which I have advocated have in the main been agreed to, no formal defence on my part is rendered necessary. I shall therefore content myself by attempting in a very few words to sum up the general results of the discussion,

to point out how far the general propositions at the end of my communication have been answered, and briefly to refer to the leading special objections to my views which have arisen during the course of the debate.

Following the order of propositions already formulated, it may be stated :

I. With regard to the clinical condition described, there seems to be a consensus of opinion, that the facts detailed are substantially correct, that the affection under consideration is of frequent occurrence, as the clinical picture is familiar to most observers, and that the course and termination of the malady is variable. One point only is questioned. Dr. Buzzard thinks that in many of these cases the cutaneous reflexes are diminished or lost. I am not prepared to deny that this is impossible—as the number of instances under observation have been too small to warrant generalisation on the question. I am satisfied, however, that this asserted condition is not essential, as in most of my cases the plantar and other skin phenomena were either normal or augmental. It may however be stated that the exact estimation and measurement of the superficial reflexes is always difficult and uncertain, and even in health they vary greatly in development, hence care has to be taken in formulating general conclusions from the behaviour of a few instances. Even supposing it to be proved that with tendinous and muscular excitability the cutaneous reflexes were depressed, it would only be further evidence that the nerve-mechanisms which preside over each of these are independent of one another.

On the question of the anatomy of this so-called hypertonic condition, as to where and in what manner the affection originates, whether it is a primary disturbance of the brain, cord, or nerves, and its prognosis, the discussion has not thrown any further light. It must be admitted that we still remain very much in the dark concerning all these important points, which eminently deserve investigation and solution, as a rational treatment for the disorder can scarcely be looked for till more is known of these elementary problems.

The only opinion hazarded as to the possible causation of this condition is by Dr. Donkin, who has been struck by the fact, that "a remarkably large number of the cases involving the legs occur in individuals who have used their limbs, either in standing or forced athletic exertion, to an inordinate extent." This functional excess or fatigue, if one, is not the only or essential cause of the malady, as it frequently occurs under circumstances which entirely preclude such an explanation.

Finally, the question as to whether the condition is due to gross organic lesion, or the result of so-called functional disorder, elicits an interesting disquisition from Dr. Hughlings-Jackson on the subject of "functional disease," with all of which I entirely concur. The expression is doubtless apt to mislead, and may be used by different persons in different senses. At the same time if a too vigorous definition of the word is not demanded, the term by common use has got to represent what is generally understood as a fairly defined class of disorders, and therefore the expression, if not strictly accurate or scientific, is convenient for want of a better. Several of the speakers, and notably Drs. Buzzard, Donkin, and Hale White, maintain that the malady under consideration may be entirely functional in nature, meaning by this that it is unattended by discoverable or permanent structural tissue change. As it is admitted that the same condition may be secondary to or the result of gross lesion, Dr. Buzzard thinks that the state of the superficial reflexes, the condition of the upper extremities, and the appearance of the fundus oculi, as he has related in his speech, give indications to enable us to differentiate the one from the other. While it would be of the utmost importance to have some reliable data to assist in the estimation between so-called functional and organic diseases, I do not think that the alleged tests are sufficiently precise or supported by enough evidence to enable us to decide the question.

II. With regard to the physiological and pathological problems in connection with muscular hypertonicity, much difference of opinion, as might be expected, has been expressed. Dr. Jackson has again ably maintained, and further explained, his views on cerebral and cerebellar influence in the causation of excessive tendon phenomena. His hypothesis seemed to receive the support of Dr. Donkin. On the other hand, Dr. Ferrier agrees with me in the belief, as I have already stated, that the physiological and pathological facts opposing this theory are so many and important, that it cannot be accepted without question. Even Dr. Jackson himself admits that there are "difficulties and objections to the hypothesis," and that there are "many facts against it." These, as well as what appears to be a more plausible explanation of the phenomena, I have indicated in my opening address. The general conclusion on this head arrived at from the discussion is, that up to the present time we have no completely satisfactory theory to explain hypertonicity of muscle and its attendant phenomena, or one against which important objections cannot be urged.

Dr. Buzzard, however, goes further, and denies that the tendon and muscle reaction to percussion has, as I have suggested, any relation to what has been termed the normal tonicity of muscle. He bases his objection on the fact that in Tabes, while the knee-jerk is absent, direct percussion on the vastus internus induces contraction of its fibres. He asks, "If the muscular 'tonus' is sufficient to allow of a vivid response of the muscle to the sudden stretching of its fibres produced by percussion of its substance, how is it that it does not suffice to permit of contraction when the sudden stretching is produced by a means which is said to be more effective than percussion?" The answer is, that in tabes the afferent elements of the reflex arc presiding over the muscle being interrupted, muscular tonicity is lost. As a result, there is loss of knee-jerk, and also loss of power of the muscle to respond to direct extensile stimulus. But as the efferent elements remain intact, voluntary motion, electrical reaction, and nutrition of both motor nerve and muscle are unaffected, and therefore, owing to ordinary neurological laws, contraction of muscle ensues when the motor nerve is struck. Accordingly in locomotor ataxy muscular contraction due to extensile stimulus, or that which results from provoked tonicity of muscle, is lost, but that due to direct irritation of the nerve, as from a blow on its substance, is preserved. In early tabes, the excitability of the motor nerves is sometimes augmented, as may be determined by electrical investigation. In such a case, even when the knee-jerk is totally abolished, a blow on the vastus internus, especially at a motor point, will cause an abnormally lively muscular contraction.

Muscular contraction following a blow on its substance may arise under other conditions, and may be due to increased irritability of the fibres themselves, which occurs when they are deprived of nervous influences, in which case the function of the motor nerves is abolished. In poliomyelitis, for example, there is loss of knee-jerk, and voluntary power, and wasting of muscle; but this last, when percussed, responds by sometimes over-activity of contraction. Here, however, the tonicity has been destroyed, and the contraction following a blow is neither the result of extensile stimulus, nor the effect of nerve percussion, but the direct product of hyper-excitability of muscular fibre.

The contraction then which follows a blow on the motor nerve in tabes, or on the muscular fibre in poliomyelitis, is in no way related to the phenomena dependent upon what has been here considered under the term muscular tonicity. The facts then which Dr. Buzzard has correctly observed and described, are in my opinion in no way opposed to the theory which has been advanced,

that the tendon phenomena are intimately connected with the reflex tonicity of muscle. It may also be concluded that percussion of a muscle may cause contraction for more than one reason; namely, from extensile stimulus, from direct irritation of motor nerve, and from direct stimulation of muscular fibre, therefore reaction following a blow does not negative the absence of tonicity. Further application of these facts seems to me satisfactorily to answer the other objections which have been raised on the same subject.

ON THE SEGMENTAL DISTRIBUTION OF SENSORY DISORDERS.[1]

BY JAMES ROSS, M.D., LL.D., F.R.C.P.,

Joint Professor of the Practice of Medicine in the Victoria University, and Senior Assistant Physician to the Manchester Royal Infirmary.

BEFORE proceeding to describe the distribution of sensory disorders in various diseases, it is necessary for us to possess a minute and accurate knowledge of the sensory nervous mechanism itself. In describing the sensory apparatus, I will presume that every one of my audience is acquainted with the knowledge imparted in the usual text-books, and I will only enter into details in dealing with that part of my subject upon which little or no information is to be obtained in ordinary anatomical works.

The sensory mechanism consists of peripheral end organs, afferent nerve-fibres and conducting-paths, and centres. The peripheral end organs vary in structure according to their situation, and according to the nature of the sensory function they subserve. Several forms are found in the skin, other forms in the muscles, and still others in the peripheral expansions of the nerves of special sense. From these end organs fibres issue which join together to form bundles that pass inwards to enter the spinal cord through the posterior roots (Fig. 1, N R), and through the homologues of these roots in the medulla oblongata, pons, and crura cerebri. On entering the spinal cord the fibres cross almost immediately to the opposite half of the cord, in which they ascend (Fig. 1, C P), and after being joined by the afferent fibres from the special senses also of the opposite side, all the strands are found lying close together in the posterior part of the internal capsule

[1] Paper read before the meeting of the Manchester Medical Society, on December 7th, 1887.

(Fig. 1, I C), and thence ascend in the corona radiata to terminate in the cortex of the hemisphere. Between their origin in the periphery and their termination in the hemisphere of the opposite side, these fibres are interrupted, more than once probably, by the interposition of grey matter. Such an interruption takes place probably in the root ganglia, and in the posterior grey horn, and some fibres may possibly be interrupted in the optic thalamus; but whether the conduction

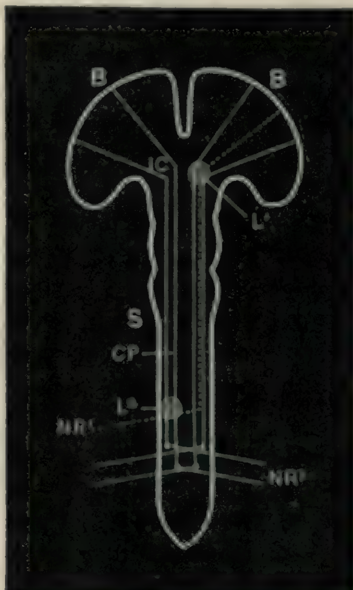


FIG. 1.—DIAGRAM OF THE SENSORY MECHANISM.

B B, Brain; S, Spinal cord; N R^1, N R^2, Posterior roots of spinal nerves; C P, Sensory conducting paths; I C, internal capsule; L^1, Lesion of internal capsule causing hemianæsthesia of opposite side; L^2, Unilateral lesion of spinal cord causing anæsthesia of opposite half of body below level, and a belt of anæsthesia in same side on level of lesion.

be maintained by continuous fibres or by relays of cells and fibres, it is unquestionable that the fibres of the afferent nerves and conducting paths find their ultimate termination in the grey matter of the cortex of the brain. But into the question of the accurate localisation of these centres I shall not enter.

Such then is a rude outline of the sensory mechanism, and now let us observe it in action. When an impression is made on the surface of the body—say the prick of a pin on the skin

—a molecular disturbance of one of the peripheral end organs or of the terminal fibrils of the sensory nerves results; this disturbance is conducted inwards by the fibres of the afferent nerves and conducting-paths until ultimately a molecular disturbance is set up in that part of the cortex of the brain which is directly connected with the injured surface, and finally the cortical disturbance becomes correlated with a feeling or sensation in the irritated part of the skin. That the essential correlate of the feeling is the molecular disturbance of the cortex is proved in two ways: *firstly*, if the disturbance set up on the suface is prevented from reaching the cortex by division of the conducting-fibres in any part of their course no feeling results; in other words, that particular part of the periphery is rendered anæsthetic; and *secondly*, when, as in the aura of many cases of epilepsy, a spontaneous disturbance begins in the sensory part of the cerebral cortex, various sensations are felt in the periphery in the absence of any outward disturbance to correspond with them. A molecular disturbance of the cortex is, then, the essential condition of consciousness, but the feeling is, in accordance with the law of eccentric projection, localised in the periphery.

Speaking broadly, every form of feeling localised in one half of the body is organised in the cortex of the cerebral hemisphere of the opposite side; and, as is well known, complete severance of the sensory conducting-paths, as they lie together in the posterior part of the internal capsule (Fig. 1, I C), causes anæsthesia of every form of sensibility of the opposite half of the body, with the exception of sight and hearing, which are only enfeebled, these organs being bilaterally associated and consequently partially organised in both hemispheres of the brain.

A unilateral division of the dorsal region of the spinal cord, (Fig. 1, L_2) by interrupting the sensory conducting paths after the afferent fibres have crossed, causes anæsthesia of the opposite half of the body below the level of the lesion, and, by injuring nerve roots, a band of anæsthesia passing round the body on the same side and on the same level with the lesion. When, however, the lesion is situated in the cervical region of the cord on a level with the roots of the brachial plexus,

the anæsthetic area will be found on the surface of the upper, and when on a level with the roots of the lumbo-sacral plexus, on the surface of the lower extremity. This statement leads me to the central problem of sensory distribution with which I wish to deal in this paper; namely, to discover the area of skin connected with each posterior nerve root, or, in other words, to find out the segmental distribution of sensory nerves.

With regard to the purely spinal nerves no difficulty arises in the case of those derived from the dorsal region of the cord, inasmuch as their well-known anatomical course sufficiently indicates their sensory and muscular distribution; but it is wholly different in the case of the nerves derived from the plexuses. An endeavour has been made to trace, by means of dissection, the fibres of each peripheral nerve up through the plexus to the root or roots from which they are derived, and this method has recently, in the hands of Dr. Herringham,[1] been attended with a considerable degree of success. I have myself for some years been engaged in unravelling the problem by means of clinical and pathological observations, and I even applied embryological considerations in a rude way to the explanation of the phenomena; but the question has been greatly advanced and placed upon a more scientific basis than previously by Dr. Paterson's[2] succession of able and instructive papers on the limb-plexuses of mammals, his investigations being wholly based upon developmental observations.

In using embryological facts for the elucidation of the distribution of cutaneous nerves in man, it is necessary for us to attend: *firstly*, to the constitution of a spinal nerve; *secondly*, to the distribution of those nerves in the lowest forms of vertebrate animals, *thirdly*, to the modifications in

[1] Herringham, (W. P.) "The Minute Anatomy of the Brachial Plexus," 'Proceedings of the Royal Society,' vol. 41, 1887, p. 423.

[2] Paterson (A. M.) 'Edin. Graduation Thesis for 1886' (gold medal); "The Morphology of the Sacral Plexus in Man," 'Journal of Anatomy and Physiology,' vol. xxi. April, 1887, p. 407; "The Limb Plexuses of Mammals," 'The Journal of Anatomy and Physiology,' vol. xxi. July, 1887, p. 611; "On the Function of the Muscle Plate and the Development of the Spinal Nerves and Limb Plexuses in Birds and Mammals," 'The Quarterly Journal of Microscopical Science,' vol. xxviii. 1887, p. 109.

the distribution of cutaneous sensory nerves which occur in the course of development, giving special attention to the *constitution of the limb plexuses*; and *fourthly*, to the distribution of the visceral sensory nerves. We shall then be in a position, *fifthly*, to apply the knowledge so gained to the explanation of sensory disorders.

1. *Constitution of a Spinal Nerve.*

A spinal nerve is derived from the cord by a posterior (superior) gangliated (Fig. 2, P r) and an anterior (inferior) non-gangliated root (Fig. 2, a r), and at the point of union of the two, the nerve

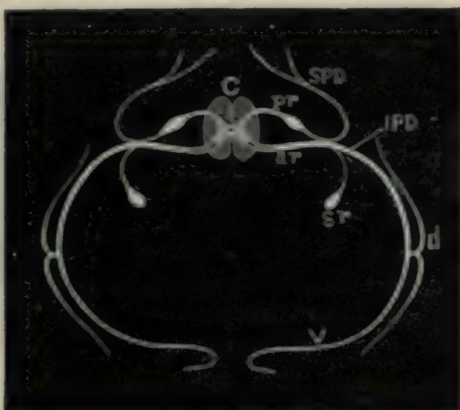


FIG. 2.—DIAGRAM SHOWING CONSTITUTION OF A SPINAL NERVE.

c, Spinal cord; P r, a r, Posterior gangliated and anterior non-gangliated root of nerve respectively; S P D, Superior primary division; I P D, Inferior primary division; d, v, its dorsal and ventral branches respectively: S r, Sympathetic (gangliated) root

divides into a superior primary division (Fig. 2, S P D) supplying the skin (the muscles do not concern us at present) of the back over the neural canal, and an inferior primary division (Fig. 2, I P D) which subdivides into a dorsal trunk (Fig. 2, d), supplying the lateral, and a ventral (Fig. 2, v) trunk supplying the ventral surfaces.

In addition to these, a branch is given off to the viscera—the sympathetic root (Fig. 2, S r)—about which we shall have more to say by-and-by.

2. *Distribution of Sensory Nerves in the Lowest Vertebrata.*

Let us now glance at the distribution of the sensory nerves in the lowest vertebrata—the amphioxus (Fig. 3) for example —and for the sake of clearness of comparison, we will assume that our typical vertebrate animal is composed of nearly the same number of segments as are found in man, and arranged in the same order, namely: three cranial (Fig. 3, S_3) (adopting the most commonly received views), seven cervical, (Fig. C_7) twelve dorsal (Fig. 3, D_{12}), and nine lumbo-sacral (Fig. 3, LS_9), the lowest two sacral and the coccygeal segments being so unimportant in man as to render it unnecessary to carry the comparison further. In such an animal each segment is supplied by its own pair of nerves.

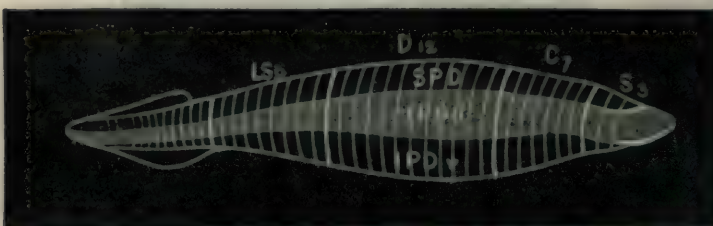


FIG. 3.—REPRESENTATION OF PRIMITIVE VERTEBRATE ANIMAL—THE AMPHIOXUS—DIVIDED FOR CONVENIENCE INTO THREE SEGMENTS FOR THE HEAD, SEVEN FOR THE NECK, TWELVE FOR THE DORSAL, NINE FOR THE LUMBO-SACRAL REGION, AND AN INDEFINITE NUMBER FOR THE COCCYGEAL REGION.

S P D. The superior primary divisions of the nerves supplying the surface over the neural canal; I P D d the dorsal trunks of the inferior primary division supplying the lateral surface of the body; and I P D v, the ventral trunk of the inferior primary division supplying the ventral surface. The parts supplied by the dorsal trunks of the inferior division are alone lined.

Now, each pair of nerves (except the last coccygeal) emerges between two segments, and if we name as in man the pair between the last cranial and the first cervical vertebra, the first cervical nerve, and the pair between the last cervical and the first dorsal vertebra, the eighth cervical nerve, it is clear that we shall have two cranial and eight cervical pairs of nerves, or ten pairs to ten segments, while in the dorsal and lumbo-sacral regions we shall have twelve and nine pairs of nerves to twelve and nine segments respectively. The shaded area in the annexed diagram (Fig. 3, I P D d) represents the

cutaneous surface supplied by the branches of the dorsal trunks of the inferior primary divisions of the nerves; the lower unshaded area (Fig. 3, I P D V) that supplied by the ventral trunks of the same division; and the upper unshaded area (Fig. 3, S P D) the part supplied by the branches of the superior primary divisions.

In the vertebrate animal, selected as our type, section of the posterior roots of a pair of nerves will cause anæsthesia of the corresponding segment of the body, and nearly the same result follows section of the posterior roots of the dorsal region in man, the chief difference being that, in the amphioxus, the nerves of one segment are not connected peripherally with adjacent nerves, whereas, in the higher animals, the nerves of successive segments anastomose freely with one another at their peripheral ends and overlap in their distribution, so that section of a pair only causes partial anæsthesia of the corresponding segment.

Speaking broadly, then, section of the posterior roots of a pair of dorsal nerves produces anæsthesia of the skin of the corresponding segment of the body, and the question we have now to determine is, how does it happen that section of the nerve roots is not attended by the same results in the other regions of the body? The answer is that the same result does follow section of nerve roots in other regions; but in them the segmental distribution is obscured because the nerves have been dragged out of their course by the displacement of old parts which occur in the course of the development of new organs, and more especially by the development of limbs. Our task now consists in giving a rough sketch of the developmental displacements which have been the main cause of diverting the nerves from their primitive segmental course.

3. *Modifications of Sensory Distribution occurring in the Highest as compared with the Lowest Vertebrata.*

The superior primary branches in man—represented in diagrams (Figs. 5 and 6, S P D) by the unshaded area which stretches from the root of the nose over the forehead, top, and back of the head, and back of the neck and body down to the tip of the coccyx—differ little in their distribution from that

of the corresponding branches in the primitive vertebrate type. The two cranial branches have united to form the first division of the fifth nerve which supplies the skin over the anterior part of the neural cavity as far forwards as the root of the nose (Fig. 6, S P D). As the neural cavity, however, has become much expanded in the higher vertebrata, and especially in man in order to make room for the greatly increased size of the brain, the skin of the back of the neck with the superior branches of the upper cervical pairs of nerves is dragged upwards in such a way that the skin over the back of the head is supplied from the cervical and not from the cranial portion of the cord, and consequently the posterior (superior) branches of the upper (anterior) cervical nerves are directed slantingly backwards and upwards (upwards and forwards) instead of backwards (vertically upwards) as in the primitive vertebrate type. From the superior branches of the upper cervical nerves we pass on to the other cervical nerves, thence to the dorsal, lumbar, sacral, and coccygeal nerves (Fig. 5, S P D) in their numerical order from above downwards (before backwards), as in the primitive vertebrate type, the chief difference being caused by the development of the erect posture in man, and consisting in the fact, that in him the nerves behind the first dorsal have an increasingly downward (backward) inclination, instead of being directed backwards (vertically) as in the primitive vertebrate animal.

In endeavouring to unravel the distribution of sensory nerves, our main difficulty arises when we come to deal with the limb plexuses. As we have just seen, the superior primary divisions do not enter into the formation of plexuses, which are consequently constituted by the complex union of branches of the inferior primary divisions of the nerves. Two suppositions are possible with regard to the plexuses; either the dorsal branches of the inferior division alone, or both the dorsal and ventral branches are arrested in the limbs.

The first alternative was maintained by Goodsir,[1] but the most cursory inspection of the distribution of the nerves, even in

[1] Goodsir, 'Edin. New Phil. Journal,' new series, vol. v. January, 1857; 'Anat. Memoirs,' vol. ii. p. 539; quoted from Paterson, 'Journal of Anat. and Physiol.' vol. xxi. p. 620.

adult man, shows that it cannot be true, at least with regard to the sensory branches. Were it true, the sensory nerves of the ventral branches would be as regular in their distribution as we have already found the sensory branches of the superior divisions of the nerves to be. Beginning with the cranial branches which have amalgamated to form the third branch of the fifth nerve, we should pass on to the cervical and then to the dorsal nerves in their numerical order. That, however, is not the order met with. The skin over the clavicle and first intercostal space is supplied by the descending branches of the cervical plexus, derived from the third and fourth cervical nerves; but, instead of the skin immediately adjoining this being supplied, as it ought to be according to Goodsir's supposition, by the ventral branches of the remaining cervical branches in their numerical order, it is supplied by the cutaneous filaments of the intercostal branch of the intercosto-humeral nerve derived from the second dorsal nerve. Clearly, then, the ventral as well as the dorsal branches of the inferior primary divisions of the fifth cervical to the first dorsal nerve inclusive are arrested in the upper limbs. From the second dorsal to the first lumbar nerve the anterior sensory branches follow in their numerical order; but we find that the skin of the inguinal and hypogastric regions, as well as the sides of the scrotum, are supplied by cutaneous branches of the first lumbar nerve, whilst the adjoining skin of the penis, the centre of the scrotum and the perinæum are supplied by the pudic, which is derived from the third and fourth sacral roots. It is manifest, therefore, that the ventral as well as the dorsal branches of the inferior primary divisions of the second lumbar to the second sacral inclusive are arrested in the lower extremity. That both branches of the inferior primary divisions of the spinal nerves enter into the formation of the limb plexuses has been still more convincingly proved by Dr. Paterson from embryological observations, but I naturally prefer to approach the subject from the clinical standpoint. It is now apparent that we cannot make a further step in unravelling the distribution of the cutaneous nerves until we study the constitution of the limb-plexuses, at any rate so far as their sensory branches are concerned.

Constitution of the Limb-plexuses.

It has been known to myself[1] for some considerable time, that if the upper extremity be placed in the embryological

Fig. 4. The lined portion on post-axial border of right arm of figure represents the area of anæsthesia caused by destructive lesion of the eighth cervical and first and second dorsal nerves, and the shaded portion in pre-axial border of left arm of figure represents the area of anæsthesia caused by destructive lesion of the fifth cervical root, as in Erb's paralysis. A back view would show a similar distribution to the front one.

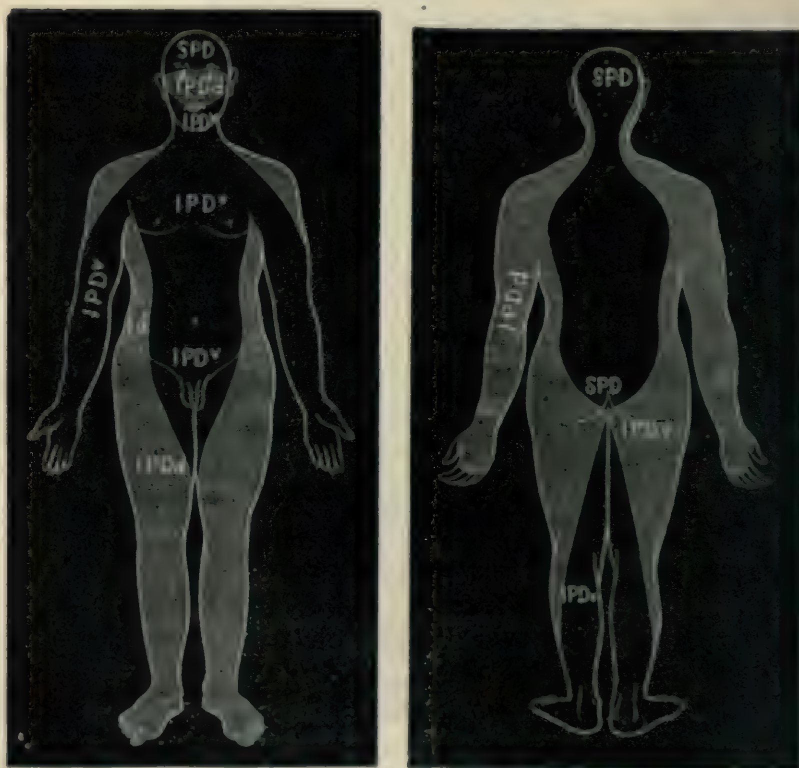
position, that is, with the thumb directed outwards or upwards and the palm forwards or towards the front of the body, the

[1] Ross (J.), "Distribution of Anæsthesia in cases of Disease of the Branches and of the Roots of the Brachial Plexus," 'Brain,' April, 1884, pt. xxv. p. 51; "A Case of Multiple Tumours, &c." *Ibid.* January, 1885, pt. xxviii. p. 501. See also Thorburn (William), "Cases of Injury to the Cervical Region of the Spinal Cord," *Ibid.* January, 1887, pt. xxxvi. p. 510.

pre-axial border from the tip of the shoulder down to the metacarpo-phalangeal articulations of the index finger and thumb is supplied by the fifth cervical root (Fig. 4, 5, 5), and that the post-axial border, from the axilla to the finger tips inclusive, is supplied by the humeral branch of the second, the first dorsal, and the eighth cervical nerves (Fig. 6, 8, 1, 2).

Observations made in cases of disease of the cauda-equina, and especially in the case of a man suffering from latent spina-bifida, which I had an opportunity of showing to the society at a previous meeting, and a report of whose case will be found in the present number of 'BRAIN,' in what will prove a very able and important paper by Dr. William Thorburn, has convinced me that, if the lower extremities be also placed in the embryological or tailor position, the pre-axial border is supplied by the cutaneous nerves of the upper four lumbar nerves, and the post-axial by the coccygeal and sacral sensory nerves. From the same case I was able to conclude, that the most distal parts of the pre-axial border were supplied from the lower of the four lumbar nerves and of the post-axial border by the higher of the sacral nerves. Dr. Herringham comes to conclusions with regard to the distribution of the cutaneous nerves of the brachial plexus which fully bear out my inferences from pathological observations. He states that (1) of two spots on the skin that which is nearer the pre-axial border tends to be supplied by the higher nerve, and (2) of two spots in the pre-axial area the lower tends to be supplied by the lower nerve, and of two spots in the post-axial area the lower tends to be supplied by the higher nerve. Dr. Paterson, who approaches the subject from a different standpoint to either Dr. Herringham or myself, formulates other important laws with regard to the distribution of the nerves in the plexuses. He finds that (1) the inferior primary divisions of the nerves entering the plexuses divide into dorsal and ventral trunks; (2) that both of these trunks are arrested in the limb; (3) that in the formation of the plexuses the dorsal unite with dorsal and the ventral with ventral branches only, and (4) that the parts derived from the dorsal parts of the embryonic limb —the extensor surfaces—are supplied by nerves derived from a union of dorsal trunk, and the parts derived from the primi-

tive ventral surfaces—the flexor surfaces—by nerves derived from a union of ventral trunks. In a recent conversation with Dr. Paterson, I find that he does not at present insist upon the truth of the third of these laws so strongly as he did when he wrote his paper on the limb-plexuses. He now admits that the small sciatic nerve, for example, is formed probably by a



FIGS. 5 and 6.—DIAGRAM SHOWING CUTANEOUS DISTRIBUTION ON THE ANTERIOR AND POSTERIOR ASPECT OF THE BODY RESPECTIVELY.

S P D, Distribution of superior primary divisions of nerves; I P D d, I P D v, Distribution of the dorsal and ventral trunks of the inferior primary divisions respectively. The area of distribution of the dorsal trunks of the inferior primary divisions is alone lined.

union of dorsal and ventral fibres, and the external or short saphenous nerve is undoubtedly so constituted.

The enunciation of these laws has now placed us in a position to map out the distribution of the lateral and anterior

branches of the cutaneous nerves. And first, with regard to the anterior or ventral branches (Figs. 6 and 7, I P D V), we have already seen that the cranial branches, distributed to the lower lip and chin, and the cervical branches, distributed to the anterior part of the neck and over the clavicle, may be taken in their numerical order. The only point worthy of notice is, that, while the branches of the superior division of the upper cervical nerves are dragged upwards (forwards) over the back of the head, the branches of the ventral trunks of the inferior division are dragged so that they slant downwards (backwards), this being rendered necessary in order to supply the skin of the upper and anterior part of the thorax, which would otherwise have been left destitute of nerves by the arrest of the lower four cervical and first dorsal nerves in the limb.

Passing on from the third and fourth cervical nerves, we come to the fifth cervical nerve, which supplies the pre-axial border of the upper extremity. Now, in the course of development, the arm rotates outward at the shoulder-joint, so that the outer and anterior surface of the shoulder slips, as it were, under the skin of the dorsal surface, and thus the dorsal branch of the inferior division supplies the skin over the ventral surface of the shoulder; while at the distal extremity of the limb there is a reverse process, insomuch as the skin of the ventral surface, with its nerves, is drawn dorsally over the last phalanx of the thumb, and the two last phalanges of the fingers. The circumflex nerve is the dorsal branch of the inferior division of the fifth nerve, while the ventral portion of the same root runs in the musculo-cutaneous nerve, the cutaneous branches of which supply the pre-axial border of the lower third of the arm and of the whole of the forearm and hand down as far as the metacarpo-phalangeal articulations of the thumb and index finger. The musculo-cutaneous nerve is also connected with the sixth root, while the median is derived from the sixth and seventh cervical, and the first dorsal roots; but it is probable that the cutaneous branches of these two nerves are derived from the fifth, sixth, and seventh cervical roots, the fibres of the first dorsal root which run in the median nerve supplying most probably the small muscles of the ball of the thumb. The anterior surface of the post-axial border of

the upper extremity is supplied by the nerve of Wrisberg, the internal cutaneous, and the terminal branches of the ulnar; these nerves are derived from the inferior cord of the brachial plexus, which, in its turn, is made up of the eighth cervical and first dorsal roots, the eighth cervical supplying, according to Herringham's law, the distal parts; that is, the ulnar half of the palm and two and a half fingers, and possibly the greater portion of the inner surface of the forearm. In tracing the anterior nerves in their numerical order, then, we pass in a descending order from the fourth cervical root supplying the skin on the anterior and inner surface of the shoulder, to the fifth root supplying the anterior surface of the pre-axial border of the lower part of the arm and of the forearm and hand, to the sixth and seventh supplying the middle of the forearm, the radial surface of the palm, and the palmar surface of the thumb and one and a half fingers; and then in an ascending order from the eighth cervical root supplying the ulnar half of the palm and two and a half fingers, and a part of the anterior and inner surface of the forearm, to the first dorsal root supplying chiefly the anterior and inner surface of the arm, to the cutaneous branches of the second root (intercosto-humeral) distributed over the second intercostal interspace; and then passing down the front of the thorax and abdomen to the symphysis pubis, we meet with the remaining dorsal nerves in their numerical order.

In endeavouring to unravel the cutaneous nerves of the lumbo-sacral plexus, it must be remembered that the limb has been rotated inwards, in the course of development, in such a way that in the adult the ventral surface is represented by the inner side and back of the thigh, the back of the leg, the heel, and the sole of the foot; while the dorsal surface is represented by the surface of the buttock, the front of the thigh over the extensor muscles, the front of the leg, and the dorsum of the foot. Following the course of the ventral branches of the inferior divisions, we pass from the anterior branch of the twelfth dorsal nerve to the hypogastric and inguinal branches of the ileo-hypogastric and ileo-inguinal nerves, both derived from the first lumbar root, the genito-crural derived from the second lumbar, the cutaneous branches

of the obturator nerve derived from the third and fourth lumbar roots, the plantar cutaneous and the internal and external plantar nerves, which are branches of the internal popliteal derived from the fifth lumbar and first and second sacral roots; and then, ascending along the outer border of the sole supplied by the external or short saphenous—a mixed dorsal and ventral nerve derived from the second and third sacral roots—we pass up the middle of the back of the leg and thigh supplied by the small sciatic nerve and its branches derived from the third and fourth sacral roots, and finally to the skin about the anus and coccyx supplied by the fifth and sixth sacral and the coccygeal nerves.

The nerves derived from the dorsal trunks (Figs. 6 and 7, I P D d) of the inferior primary divisions are distributed (the area of their distribution is alone shaded in Figs. 6 and 7) in a similar manner to the nerves of the ventral trunks, only in tracing them we must pass down the dorsal instead of the ventral aspects of the limbs. The two cranial dorsal trunks unite to form the second branch of the trigeminus which is distributed to the upper lips, nose, and side of the cheeks, and from these we pass down the side of the neck, taking the lateral branches of the four upper cervical nerves in their numerical order. From the tip of the shoulder supplied by the fourth root we pass to the dorsal branch—the circumflex nerve—of the fifth root, thence to the external and internal cutaneous and the radial nerves, which are branches of the musculo-spiral, derived chiefly from the sixth and seventh roots; now ascending along the posterior aspect of the post-axial border, we encounter the posterior cutaneous branches of the ulnar, the internal cutaneous, the nerve of Wrisberg, and the humeral branch of the intercosto-humeral, derived respectively from the eighth cervical and first and second dorsal roots, and then we descend along the lateral aspect of the trunk, taking the remaining dorsal nerve-roots in their numerical order.

Passing now to the lumbar roots we come to the iliac branches of the ileo-hypogastric, ileo-inguinal nerves, derived from the first lumbar, the external cutaneous from the second, the middle and internal cutaneous and the long or internal

saphenous branches of the anterior crural derived from the second, third, and fourth lumbar roots; and now crossing over the dorsum of the foot and ascending along its external border to the outer surface of the leg we come in succession to the cutaneous branch of the musculo-cutaneous, the external or short saphenous, and the cutaneous branch of the external popliteal, all derived from the fifth lumbar and the first, second, and third sacral roots; and then we pass along the external aspect of the back of the leg and thigh, which is supplied by the small sciatic nerve, derived from the third and fourth sacral roots, and finally we come to the skin of the anus and perinæum, middle of the scrotum, and penis, supplied by the inferior hæmorrhoidal, inferior pudendal, and pudic nerves, derived from the third and fourth sacral nerves, and to the skin about the anus and coccyx, supplied by the coccygeal nerves. I have, however, been unable to discover how the dorsal, as apart from the ventral branches of the lower sacral and coccygeal nerves, are distributed.

4. *Distribution of the Visceral Sensory Nerves.*

Having now mapped out the cutaneous surface of the body into its segmental nerve-territories, it remains for us to deal, as briefly as possible, in the same way with the innervation of the viscera. In describing the constitution of a spinal nerve allusion was made to the sympathetic branch, which parts from the nerve at or near the union of the posterior (superior) and anterior (inferior) roots. These branches form the *rami communicantes*, which, emerging from the spinal cord along with the other roots, enter the lateral and collateral ganglia of the sympathetic, ranged along the sides and front of the spinal column. Now the researches of Gaskell[1] have shown that there are two varieties of these branches; in the one the fibres are destitute of medulla, while in the other they are medullated, the two forming respectively a grey and white trunk. Gaskell has also shown that the grey trunk is made

[1] Gaskell (W. H.), "On the Structure, Distribution of, and Functions of the Nerves which innervate the Viscera and Vascular Systems," 'The Journal of Physiology, vol. vii. 1886, p. 1.

up of nerve fibres which have a centripetal course; they have already passed through the ganglia of the sympathetic, where they have been deprived of their medulla, and now pass inwards to supply the blood-vessels of the vertebral column, and the cord with its membranes. According to this view, the white *rami* alone contain fibres for the innervation of the viscera, or *splanchnic* fibres. The splanchnic nerves issue, according to Gaskell, from the spinal cord in definite thoracic, cervico-cranial, and sacral regions. The thoracic group emerge with the roots of the spinal nerves from the second dorsal to the second lumbar inclusive. The cervico-cranial group are contained in the internal branch of the spinal accessory nerve, which is derived from the first, second, and probably the third cervical nerves, and its fibres pass out to join the ganglia on the main stems of the vagus and glosso-pharyngeal nerves. The sacral group, which constitute the main portion of the *nervi erigentes*, emerge along with the roots of the second and third sacral nerves, and pass directly to the hypogastric plexus; whence they send branches upwards to the inferior mesenteric plexus and downwards to the bladder, rectum, and generative organs.

The question now is, from what cells in the spinal cord do these fibres take their origin? Some years ago, I had myself suggested that the sympathetic fibres were derived from the cells of Clarke's column. My reasons for coming to this conclusion were that the ganglion cells in Clarke's column are bipolar and in other morphological respects similar to the cells of the sympathetic ganglia;[1] that this column is present in the thoracic portion of the cord, which supplies nerves to the viscera; that it is absent on a level with the lower cervical and lumbo-sacral portions which supply nerves to the limbs; and that one of the nuclei of origin of the spinal accessory nerve and of the vagus—the great visceral nerve—consists of a group of bipolar cells like those of Clarke's column. This idea has, quite independently of anything that I have said, been worked out in detail by Dr. Gaskell, and placed by him

[1] This idea was, I believe, first suggested to me by a passage in Poincaré's work on the Nervous System, which I have not had time or opportunity to identify, otherwise I should have given the distinct reference.

upon a scientific foundation. He has also shown that some splanchnic fibres are derived from the cells of the lateral part of the anterior grey horn—the respiratory tract of Bell; while others are connected with the spinal ganglia and enter the cord in the posterior roots, these being doubtless endowed with sensory functions. It has not, however, been found possible to separate the sensory from the motor fibres in their peripheral distribution, but it is highly probably that the two kinds of fibres run together in the same nerves to reach the respective viscera. I shall therefore presume upon the knowledge of my audience of the further distribution and connections of the splanchnic nerves, including the *vagi*, and shall now proceed to apply the distribution of sensory nerves just sketched out to the explanation of the sensory disorders met with in various diseases.

5. *Application of the Distribution of Sensory Nerves to the explanation of Sensory Disorders.*

In dealing with this part of my subject, I shall pass over the more familiar applications of sensory distribution in the diagnosis of disease; such as the indication which the presence of anæsthetic and hyperæsthetic belts of skin surrounding the body and passing down the limbs gives for localising disease in the spinal cord and spinal roots, and shall direct my attention to the explanation of the pains observed in disease of the internal organs. Now, disease of an internal organ—say the *stomach*—is accompanied by pain over the seat of the organ—the epigastrium in the case of the stomach—a pain that may be regarded as of splanchnic origin and named accordingly the *splanchnic* pain. In addition to this pain, the patient complains of pain between the shoulders and in front of the chest. On being asked to point to the seats of these pains, he places the palm of the hand broadly across the chest just below mid-sternum for the front pain, and indicates the shoulder pain by passing one hand over the shoulder of the same side and pointing with the tips of the fingers to a situation somewhat low down between the shoulders, moving the finger tips from side to side to show that the pain is

bilateral and somewhat diffused. Short, stout patients fail to reach as low down between the shoulders as they desire by passing the hand from above, and then attempt to reach it by passing the forearm behind the back and directing the tips of the fingers upwards; most of them find themselves still further now from the desired point than before, and immediately return to the first means of indicating the seat of the pain by passing the hand over the shoulder. Some patients indicate the seat of the pain by passing the forearm behind the back, flexing and supinating the forearm so that its radial border rests on the shoulder blade, and then moving the nail of the thumb transversely across the spine from side to side opposite the spinous process of the fourth or fifth dorsal vertebra. These associated pains of dyspepsia are, therefore, situated in the region of distribution of the posterior and anterior branches of the fourth and fifth dorsal nerves. Sometimes the patient may experience a feeling of oppression or constriction in the left side, which may be caused either by irritation of the lateral branches of the same nerve, or by a partial spasm of the intercostal muscles supplied by them. The explanation of these associated pains in dyspepsia is to be found in the fact, that the stomach has been developed as an upper thoracic organ, and that in its downward displacement it has carried its nerves with it. The splanchnic nerves of the stomach are derived from the fourth and fifth, and probably the sixth dorsal nerves, and when the splanchnic peripheral terminations of these nerves are irritated the irritation is conducted to the posterior roots of the nerves, and on reaching the grey matter of the posterior horns it diffuses to the roots of the corresponding somatic nerves and thus causes an associated pain in the territory of distribution of these nerves, which may appropriately be named the *somatic* pain. Another very important associated pain of gastric disorder is brow-ache. The patient indicates the seat of this pain by placing the palm of the hand broadly across the forehead, and while raising the hand he generally bends the head a little forward and to the side to meet the palm. The severity of this pain is indicated by the mournful and lugubrious expression of the patient while the hand is being placed on the forehead. It is most likely that this pain is caused by irrita-

tion of the pneumogastric nerve, but as what we may call the somatic associated district of this nerve extends over most of the head and face it is difficult to know why the forehead should be particularly selected. It has occurred to me that the pain might be due to some changes in the cerebral vessels caused by irritation of the vaso-motor nerves of the carotid cerebral circulation, which are derived from the upper thoracic region, but this view does not afford a very satisfactory explanation.

Let us now consider the associated pains of disease of the *lungs*. Disease in the substance of the lung, which does not press upon neighbouring parts, is not accompanied by very definite pains of any kind, unless the pleura be implicated. In uncomplicated cases of pneumonia the patient complains of a dull pain in the region of the inflamed organ, which is most probably of splanchnic origin; while the most prominent associated disorders appear to be caused by irritation of the pneumogastric nerves. These consist of palpitation, gastric disorders, and rumbling of the bowels with an uneasy feeling of flatulence. An attack of acute crupous pneumonia, especially in young people, is often ushered in by severe vomiting, accompanied occasionally by diarrhœa, so that at first the general symptoms are more like those of acute gastric catarrh than of an inflammation of the lungs. In the course of a pneumonia, also, patients often complain of a pain at mid-sternum and between the shoulders, such as we have found to accompany primary disease of the stomach, and they sometimes appeal to the medical attendant to prescribe something to remove the wind on the stomach, the presence of which is also indicated by belchings.

The pain of *pleurisy* is often felt over the anterior part of the abdomen, this being clearly caused by direct irritation of the intercostal nerves in their passage over the pleura in the first part of their course. The accompanying stitch of pleurisy is mainly caused by spasm of the intercostal muscles and of the diaphragm from reflex irritation. All these pains are, therefore, the result of direct or reflex irritation of the somatic nerves, which pass in or near the inflamed membrane. There is, however, an associated pain

often present in pleurisy for which it is somewhat difficult to find an explanation. We allude to pain, often severe and urgent, over the outer third of the clavicle, and reaching to the shoulder tip—the territory of the anterior branch of the fourth nerve. Sometimes the pain feels as if a nail were being driven into the joint, and in these cases it may be inferred that the sensory nerves of the joint (a branch of the suprascapular nerves derived from the fourth and of the circumflex nerve derived from the fifth cervical root) are in a state of irritation. Now the fourth and fifth cervical pairs of nerves have no splanchnic connections, and consequently this pain cannot be the associated somatic pain of splanchnic irritation. In observing a case of pleurisy a few days ago, and casting about for an explanation, one of the students suggested that, considering the connections of the phrenic, the pain might be caused by irritation of that nerve. It then occurred to me that Peter[1] had described a phrenic neuralgia, and on referring to his description I found that he regards pain over the shoulder tip as a constant symptom. It is only right to add, that the phrenic nerve is regarded by some physiologists as a purely motor nerve; but, considering how exquisitely sensitive the diaphragm becomes in pleurisy and in peritonitis, it can hardly be destitute of sensory nerves. In favour of the view, that the shoulder tip pain is caused by irritation of the phrenic, is the fact that essentially the same pain is met with in pericarditis, peritonitis, abscess of the liver, and during the passage of gall stones; while we shall immediately see, that it is probably met with in a more or less disguised form during attacks of angina pectoris.

A patient, suffering from pleuro-pneumonia, attended by me at present, along with my friend Dr. Judson Bury, and who has also been seen several times by Sir W. Roberts, manifests the pains of the two diseases in a very obvious manner. He is a highly nervous man, of gouty constitution, and as in previous illnesses he has proved intolerant of opium in any form, it is found most difficult to allay his sufferings, so that a very undesired opportunity is afforded for studying

[1] Peter (M.), "Neuralgie diaphragmatique et faits morbides connexes." Archives Générales de Médecine, vi^e série, tome xvii., 1871, p. 303.

the character of his pains. A physical examination reveals crepitation and dulness of the bases of both lungs; but the disease predominates on the right side, and the pleuritic stitch is entirely limited to it. When the patient is quiet and freest from pain, he suffers only from a dull aching on the right side over the posterior and lateral aspects of the lower part of the chest, which is most likely the splanchnic pulmonary pain. In addition, he complains of pain at mid-sternum, and of a feeling of flatulent distension of the stomach and bowels, which is likewise indicated by occasional belching, and by borborygmi and the passage of wind *per anum*. These periods of comparative calm are of short duration, being interrupted by paroxysms of cutting and radiating pains, neuralgic in character, and of great severity. "When the attack comes on," says the patient, "I feel a cutting pain in the right side which stops my breathing; my heart then begins to jump; and," placing his hand over mid-sternum, he continued, "I have a severe pain here which I think would be relieved if I could only get up some wind." The description of the remaining pains was only elicited by cross-examination. On my asking him if he had a trilling pain along the outer third of the clavicles passing to the shoulder-tips, his countenance immediately lighted up with intelligence, and he said, "I have *that*," and evidently referring to my description, he continued, "that is just it exactly." He then indicated by his finger-tips that the shoulder-pains passed down for about two inches along the outer aspects of the arms. Curiously the pain is more persistent in the left than in the right shoulder, although the pleurisy is situated on the right side, and there are no signs of pericarditis and no suspicion of aneurism. Wishing to know whether or not he had the pain between the shoulder-blades which so frequently accompanies the mid-sternal pain in gastric disorder, I passed my hand to that situation, and asked him if he had a pain there, "No," he said, "it is at the back of my neck." To my surprise I found that the situation of this pain was the nape of the neck, and so urgent had it been a few hours before my examination that he got the nurse to apply a hot linseed-meal poultice to the nape, which he said afforded him great

relief. As this pain did not extend up the occiput, it was situated in the surface area supplied by the posterior branches of the fourth and fifth cervical nerves, and it is the lateral and anterior branches of these same nerves which supply the skin over the outer part of the clavicles and shoulders, and consequently both pains are likely to have been caused by irritation of the phrenic nerve. At the beginning of a paroxysm of pain the action of the heart becomes tumultuous and irregular, and, although the patient is a stout man, the abdominal aorta is seen and felt to pulsate strongly. The bowels also rumble a good deal, and he seems to find some relief in belching, and still greater when wind passes *per anum;* these last symptoms being probably due to associated irritation of the pneumogastric nerves.

The associated pains of *cardiac* disease are best studied during attacks of angina pectoris, although they may be present in diseases of the heart and large blood vessels unaccompanied by anginal paroxysms. Some years ago Dr. Allen Sturge,[1] in a very able paper which does not appear to have attracted so much attention as it ought to have done, and the importance of which had escaped my own notice until my views on this subject had already been formed, endeavoured to show that the sensory phenomena of angina pectoris were caused by a primary irritation of the peripheral ends of the cardiac sympathetic nerves, which on reaching the spinal cord became diffused in the grey matter of the posterior horns, thus causing the pains which shoot between the shoulders and down the arm. After discussing several other hypotheses, Dr. Sturge asks, "Does the spinal cord offer a more probable solution to the problem?" In reply to this question he says, "The cardiac nerves of the sympathetic come from the three cervical ganglia on both sides. Of these, the largest nerves are the two nerves which come from the middle cervical ganglia. The strands passing from the ganglia to the spinal cord pass in the trunk of the fifth and sixth cervical nerves; those passing from the inferior cervical ganglia, in the trunk of the seventh and eighth cervical nerves. It is these four nerves

[1] Sturge (W. Allen), "The Phenomena of Angina Pectoris and their bearing upon the Theory of Counter-irritation," 'BRAIN,' Vol. V. January, 1883, p. 492.

which, in conjunction with the first dorsal, form the brachial plexus. We thus see that the region of the spinal cord which gives origin to the brachial plexus gives origin also to the greater part of the fibres which eventually find their way to the heart. Wherever, then, the original commotion may have taken place in an attack of angina pectoris, whether in the cardiac ganglia of the sympathetic, or in the spinal cord, and whether the commotion be due to some peripheral irritation from disease of the heart's substance, or be a spontaneous outburst on the part of the nerve cells implicated, it is evident that it is only when the commotion has begun in the cord, or has passed up to the grey matter of the spinal cord from the sympathetic, that any great extension in its area can take place, such as that of which I am speaking." In this remarkable passage Dr. Sturge has clearly anticipated, in principle at least, the view advanced in these pages; although our knowledge of the distribution of sensory nerves at the time at which he wrote, was much too vague to enable him to map out accurately the area affected. A similar idea also has been well expressed by Dr. Allbutt,[1] who says, "As in angina pectoris and the pseudo-angina, so in gastralgia, the spinal nerves may be included in the paroxysms, or may take even a chief part in them, the visceral and overlying spinal nerves being grouped in function and in suffering together." According to Gaskell's observations the heart derives its splanchnic nerves from the second dorsal pair of nerves, and we must therefore expect that the central area of the associated pains of cardiac disease will be found in the region of distribution of these nerves. In some attacks, however, the pains will be likely to become diffused in the areas of distribution of the first dorsal and eighth cervical above, and in those of the third and fourth dorsal below, and may very possibly extend on the one hand to higher, and on the other to lower nerves than these, in aggravated cases. Some weeks ago I obtained a very graphic description of the cardiac pains from a patient in the Infirmary under the care of my colleague, Dr. Simpson, who was suffering from free aortic regurgitation

[1] Allbutt (T. Clifford), "On Visceral Neuroses, being the Gulstonian Lectures for 1884;" London, 1884, p. 31.

and dilatation of the left ventricle, accompanied by severe attacks of angina. On being asked to describe his sensations during these attacks, he said, without any prompting, "My pain starts here"—pointing with the tip of the middle finger of the left hand to a point a little below mid-sternum; "it then shoots back between my shoulders"—moving the tips of the fingers of the right hand across the spine on a line with the second dorsal vertebra—"and darts down the inside of my arm to the elbows"—at the same time drawing the backs of the fingers of the right hand from the left axilla down the inner side of the arm to the elbow, and repeating a similar movement with the left hand down the inner side of the right arm. He then raised both hands and placed the tips of the fingers of each hand—the hands being held nearly at right angles to the body—over the second ribs, below the middle of the clavicles, and said, "I have a feeling of great tightness here." The sensory distribution of the second dorsal nerves could hardly have been better mapped out by an instructed anatomist than it was by this patient, the tightness over the second rib being most probably caused by spasm of the second, and probably also of the first intercostal muscles. A second case of angina came under my observation a short time ago, in which the patient said that the pain shot across his back and down the back of the left arm, and, on being asked to indicate the seat of the arm pain, he passed the right hand over the left arm below the shoulder, and moved the tips of the fingers along the post-axial border of the arm from the axilla to the elbow—the area of distribution of the dorsal branches of the second and first dorsal roots. But the associated pains of angina pectoris are not always so strictly limited as they were in these cases. Sometimes the pains pass down the inner side of the forearm to the tips of the ring and little fingers, and in these cases we must assume, that the irritation has extended from the roots of the second to those of the first dorsal and eighth cervical nerves. In other cases, I believe that the pain passes down the outside of the arm, in the area of distribution of the fourth and fifth cervical nerves, and as these nerves have no splanchnic connections, we must assume that the pain in this situation is caused by irritation of

the phrenics, which, in its turn, is produced by an associated spasm of the diaphragm. The shoulder-tip pain forms a marked feature in many cases of acute pericarditis, and Dr. Judson Bury informs me that this disease in young people is, like pneumonia, frequently ushered in by severe vomiting. In a case of aneurism of the aorta now in the Infirmary, under the care of my colleague, Dr. Leech, the patient complains of a pain shooting across the spine and shoulder-blades on a level with the third or fourth dorsal vertebra. There is no reason to believe that the posterior branches of the third or fourth dorsal nerves are subjected to direct pressure or irritation, and therefore the pain must be an associated one, caused by irritation of the sympathetic roots of these nerves, which are known to supply splanchnic fibres to the large blood-vessels. Anginal attacks, and, indeed, all diseases attended by great anæmia of the brain, are accompanied by a feeling of weight and oppression of the top of the head, but of the causation of this pain I cannot give a satisfactory account.

Let us now turn to the great organ which lies in the cavity of the skull—the *cerebrum*. The associated pains of cerebral disease are variable, and we are not able to give a satisfactory explanation of them. In organic diseases of the brain, such as inflammation and tumours, the pains are very variable in their intensity, and may be frontal, temporal, occipital, vertical, or diffused in their localisation. In exhausted conditions of the brain, however, a disagreeable sensation is felt over the back of the head. It is sometimes described as a numbness; at other times the skin is said to be over-sensitive, and patients find the combing and brushing of the hair a very disagreeable and often a painful process. One patient said that "the pain seems as if some one were pulling my hair from the roots," and I have known several young girls to have their hair cut short in order to avoid the necessity of combing long hair. This pain is aggravated by a slight touch, and often soothed by deep and steady pressure, and consequently it must surely be caused by irritation of the posterior branches of the upper occipital nerves; but what is the source of the irritation, and by what channels it is conducted to the spinal cord, I am

unable to say. Why irritation of the branches of the pneumogastric fibres to the stomach should give rise to brow-ache, anæmia of the brain to a feeling of pressure on the vertex, and exhaustion of the nervous system to this occipital tenderness, I cannot tell, but I have abundant clinical evidence of the fact. In reference to the last pain, it must be remembered that the cervico-cranial splanchnic region is situated as low as the three upper cervical nerves.

Passing now to the *liver*, the splanchnic pain consists of a feeling of fulness in the right hypo-chondriac region over the seat of the organ; while the somatic pain is referred to the inferior angle of the scapula and sometimes across the spine to the same point on the left side, these being the regions of distribution of the posterior branches of the seventh and eighth nerves from which the splanchnic nerves of the organ are most probably derived. In abscess of the liver, perihepatitis, and all diseases which are situated in or near the upper surface of the organ, the shoulder-blade pain is often accompanied by the shoulder-tip pain of phrenic irritation. During attacks of gall stones, the splanchnic pain is felt over the distended organ, and the associated pains as a rule shoot to the back and upwards. In exceptional cases, of which a probable example came under my observation a few days ago, the associated pain is felt towards the epigastric region, and in these cases it is probable that a calculus has been arrested at the point of entrance of the common duct in the duodenum.

In diseases of the *bowels* the splanchnic pain may be variously situated, but the associated somatic pains are felt in the back in the area of distribution of the posterior branches of the tenth and eleventh dorsal nerves, and in the front about the umbilicus. Sir James Sawyer[1] has recently pointed out that certain patients, with digestive disorders, complain of pain in the back, the situation of which they indicate by passing the extended thumb across the back at a point midway the scapular pains of liver disease and the loin pain of kidney disease. He believes that it is caused by a loaded colon. I know the cases well, and attribute the pain to irritation

[1] Sawyer (James), "Note on the Cause and Cure of a Form of Backache;" 'Lancet,' January 1st, 1887, p. 17.

of the branches of the inferior mesenteric plexus, derived chiefly from the eleventh and twelfth dorsal roots.

Diseases of the *spleen* are probably not accompanied by marked associated somatic pains.

The lumbar organs consist of the kidneys, with the ureters, the testicles, and the ovaries. The *kidneys* have not undergone much displacement during development and consequently the splanchnic and somatic pains coincide, the pains consisting of a dull aching in the loins. When, however, the pelvis and ureter become implicated, the associated pains shoot down the inside of the thigh, in the region of the ileo-inguinal and genito-crural nerves, derived from the first and second lumbar, and along the lateral aspect of the thigh, in the territory of the external cutaneous nerves derived from the second lumbar root. These pains are also often accompanied by spasm of the cremaster muscle.

Disease of the *testicle*, in addition to the splanchnic pain in the organ itself, is accompanied by dragging pains in the loins and by pains shooting down along the inguinal branch of the ileo-inguinal nerve.

In disease of the *ovaries* a woman indicates the situation of the pain in the back by placing the tips of both hands over the lower part of the lumbar region, just above the iliac crest on each side, the region of distribution of the posterior branches of the second lumbar nerves, and in front by placing the ulnar border of the hands a little above the groins, the region of distribution of the inguinal branches of the ileo-inguinal nerves, but in front it is difficult to separate the splanchnic and somatic pains. The associated pains are sometimes referred to the iliac region and hip-joints, parts which receive their sensory innervation from the three upper lumbar nerves, and then the patient indicates the seat of the pain by placing the palm of her hand in a slanting direction over the iliac region above the hip.

The pelvic organs consist of the bladder, rectum, and uterus. These organs are supplied by the sacral splanchnics, derived from the second and third sacral roots.

The splanchnic pain of *bladder* disease is a gnawing pain in the hypogastric region over the organ, while the associated

somatic pain passes along the urethra to the tip of the penis parts supplied by the pudic nerve, derived from the third sacral nerve.

The pains of *rectal* disease consist of straining and tenesmus, which are partly caused by muscular spasm and partly by irritation of the inferior hæmorrhoidal nerves; the associated disorders consist of pains in the urethra, in the course of the pudic nerve. In some cases pains shoot down the back of the thigh in the course of the small sciatic nerve, derived from the third sacral root.

Disease of the *uterus*, apart from the ovarian irritation with which it is so generally accompanied, is declared by a splanchnic pain in the hypogastric region, and by a somatic associated pain over the lower part of the sacrum, the seat of which the patient indicates by placing the back of the hand broadly across the sacrum, in the region of distribution of the posterior branches of the second, third, and fourth sacral nerves. Ulcerations of the *os uteri* are frequently accompanied by coccygeal neuralgia, the irritation probably extending from the splanchnic connection with the third sacral nerve, In uterine displacements and enlargements, many of the accompanying pains are caused by direct irritation of the sacral and coccygeal nerves as the result of pressure.

Did time permit, I should like to have pointed out the light the theory I have advanced sheds upon the motor, vaso-motor, and secretory disturbances which usually accompany visceral pains, and upon the associations and successions of these sensory disorders, which, like the auræ of epileptic seizures, owe their origin to spontaneous discharges of energy from the cortex of the cerebrum. I must, however, come to a conclusion; but, before sitting down, I desire to tender my thanks to Professor Young for much assistance in tracing out the course of the nerves, and for the diagrams which illustrate the text. I am also indebted to Dr. Paterson for much useful anatomical and embryological information.

EXPERIMENTS ON SPECIAL SENSE LOCALISATIONS IN THE CORTEX CEREBRI OF THE MONKEY.[1]

BY E. A. SCHÄFER, F.R.S.,

Jodrell Professor of Physiology in University College, London.

In the long series of experiments upon the monkey's brain with which I was engaged in conjunction with Mr. Victor Horsley during more than two years,[2] we obtained as the result of lesions of certain parts of the cortex indications, more or less marked and permanent, of the partial or complete abolition of certain special sense functions, while others remained to all appearance intact, no matter what portion of the cortex might be implicated in the lesion. The sensory impressions, the perception of which was thus interfered with, were those of sight and touch, and associated with loss of the latter was impairment of general sensibility. On the other hand, we did not obtain in any of our experiments unmistakable evidence, nor indeed any evidence at all, of the impairment or abolition of the senses of hearing, smell, or taste.

With regard to vision, our experiments were not conclusive. We found that extensive lesions, both of the occipital lobe and of the temporal lobe, were invariably followed by visual disturbances, taking the form, when the operation was confined to one side of the brain, of bilateral homonymous hemianopsia; but in nearly every case the hemianopsia was merely temporary, and after a certain time we could not in

[1] The experiments to which this article relates have, for the most part, been performed in conjunction with Dr. Sanger Brown, of Chicago. The details of our observations, with representations showing the exact post-mortem condition of the brains operated on, are given in a paper submitted by us to the Royal Society, which was read at the meeting of that Society on December 16th, 1887.

[2] Horsley and Schäfer, "A Record of Experiments upon the Functions of the Cerebral Cortex," 'Philosophical Transactions,' 1887.

our monkeys obtain any distinct evidence of the persistence of the visual defect. The most marked results of this kind were obtained when the occipital lobes were the seat of the operation, extensive unilateral lesions in this region producing hemiopia, and bilateral lesions producing amblyopia; but in neither case were the symptoms permanent, and after a time the animals, so far as we were able to determine, could see as well as their intact fellows. In one case only did the hemiopia persist, and this was one in which, after a bilateral lesion of both occipital lobes had been carried out and the temporary blindness thereby produced had been recovered from, the angular gyrus of one side was destroyed. This second operation, made upon an animal in which the occipitals had already been extensively destroyed *without* permanent blindness, did produce a condition of hemianopsia which lasted until the animal's death some three months later. We were of opinion, at the time, that this instance might warrant us in taking up a position similar to that of Luciani and Tamburini,[1] and intermediate between those of Ferrier and Munk—the former of whom originally denied the participation of the occipital lobe in the visual perceptive function, and still appears to regard it as subordinate to the angular gyrus; whereas the latter would localise those perceptions entirely in the occipital lobe, and deny all participation of the angular gyrus. But we made only four experiments upon these regions, and in none of them was the removal of the occipital lobe complete, as was proved by post-mortem examination of the brains. They were not, therefore, decisive against Munk's statement, that persistent hemiopia or blindness follows extirpation of one or both occipital lobes alone, and it became necessary to pursue further enquiries in order to test its accuracy.

This I have now accordingly done, in conjunction with Dr. Sanger Brown. With reference to visual perceptions, we have experimented both upon the angular gyri and upon the occipital lobes.

In one monkey, a small, active and intelligent Rhœsus, we

[1] Luciani and Tamburini, "Sulle funzioni del cervello; seconda communicazione," 'Centri psico-sensori corticali,' 1879. (Abstract in 'Brain,' Vol. II.)

destroyed, as completely as we could from the surface by the actual cautery, the grey matter of one angular gyrus. We tested the sight immediately after complete recovery from the anæsthetic, but could discover no defect of vision, nor any loss of movement of the eyes or eyelids, nor any anæsthesia of the cornea or conjunctiva. When the eye of the same side was closed by plaister, the animal continued to see perfectly well with the eye of the other side, nor could we discover any diminution in the visual field.

The same tests were applied, from time to time, during the few days succeeding the operation; and as completely negative results were invariably obtained, we proceeded to destroy in the same manner, a week after the first operation, the angular gyrus of the opposite side. This lesion also was not followed, either immediately or at any subsequent time, by any visual defect that we could discover by the most careful tests we knew how to apply. That the lesion was complete on both sides was proved by the post-mortem examination, which was made some months later (see Fig. 1). Here, then, was a

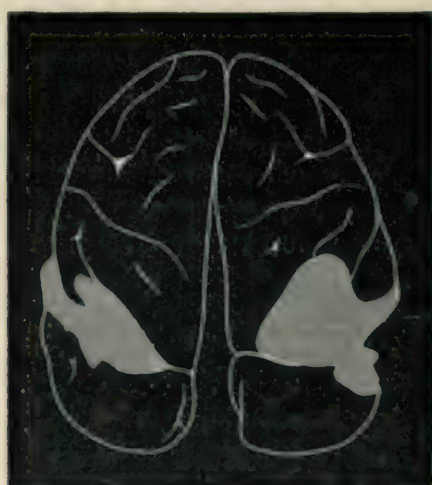


FIG. 1.—Brain of a Monkey, with complete destruction of both angular gyri. Viewed from above.

monkey with total absence of the cortex of both angular gyri, without exhibiting at any time any appreciable defect either in

its visual perceptions or its ocular movements, or in the sensibility of the globe of the eye. This animal was kept for several months, was examined carefully not only by ourselves but by many other people, and was exhibited to the Neurological Society at its meeting in this laboratory last winter.

A single well-marked negative case like this is conclusive against the idea, that in the monkey cerebral visual perceptions are *localised* in the angular gyri. As already stated, this idea was formerly entertained by Dr. Ferrier;[1] but it rested, apart from general analogies, chiefly upon experiments which were at that time, before the days of antiseptic surgery, necessarily somewhat rough. In consequence of his more recent experiments, undertaken in conjunction with Professor Gerald Yeo,[2] Dr. Ferrier has somewhat modified his earlier conclusions. In the 'General Results' of their experiments on the occipito-angular region (*op. cit.* p. 504) we find the following statement:—

"Complete destruction of the angular gyri on both sides causes for a time[3] total blindness, succeeded by lasting[4] visual impairment in both eyes. Destruction of the convex aspect of the angular gyrus on one side causes temporary abolition or impairment of vision in the opposite eye. The defect is not of a hemiopic character. Subsequent similar lesion of the other angular gyrus causes bilateral visual defect, also only of transient duration."

And elsewhere[5] Dr. Ferrier gives it as his mature opinion, that "the angular gyrus is the special region of clear or central vision of the opposite eye, and perhaps to some extent also of the eye on the same side."

These statements and opinions are not consonant with our results in the case of the monkey we have above described. Even half an hour after the operation on either side we obtained no evidence whatever of defective vision, the animal being at the time quite lively and having recovered from the

[1] See the first edition of his book on the 'Functions of the Brain.'

[2] Ferrier and Yeo, "On the effects of the lesion of different regions of the Cerebral Hemispheres," 'Philosophical Transactions,' 1884

[3] Three days, *op. cit.* pp. 493, 494.

[4] Observed, however, for one month only, p. 494.

[5] 'Functions of the Brain,' 2nd edition, 1886, p. 288.

chloroform. It is true we did not simultaneously remove both angular gyri, and could not therefore, according to Dr. Ferrier's results, expect to get total blindness even immediately after the destruction of the second gyrus. But we should in each case have obtained temporary blindness of the opposite eye (not hemiopia), whereas we got no appreciable result whatever. Since, however, it might be objected, that although we destroyed the cortex of the angular gyrus right up to the fissures bounding the gyrus, we had still left that at the bottom of these fissures, we determined in another animal to make a complete removal of the gyrus angularis in its whole depth and extent. We accordingly separated the lips of the fissures, and scooped away the entire gyrus angularis of one side, producing thereby a gap in the surface of the brain of considerable depth. This operation *was* followed by a disturbance of visual perceptions; but the disturbance was not amblyopic; it was distinctly hemiopic. The condition lasted for a few days, gradually passing off, leaving vision unimpaired. The result, although somewhat different from that which we obtained from the destruction by cauterisation of the exposed grey matter, by no means corresponds to Dr. Ferrier's results, nor does it fit in with his conclusions; and, as will presently appear from our experiments upon the occipital lobe, it is susceptible of an interpretation which would exclude the angular gyrus from participation in the function of cerebral visual perception, for the result can be explained by the vascular disturbance which is produced in that lobe by so radical a removal of the neighbouring gyrus.

We may remark incidentally, that neither this case nor the other one with (successive) double extirpation of the gyrus angularis offers any support to the statement of Professor H. Munk, that the convolution in question is related to the sensibility of the opposite eyeball and concerned in regulating its movements.[1] On this point we entirely agree with Dr. Ferrier, who states that he has "never seen, either on unilateral or bilateral destruction, the slightest appearance of ptosis or paralysis of the ocular muscles," and has "found the sensibility

[1] H. Munk, "Ueber die Functionen der Grosshirnrinde, Gesammelte Mittheilungen," 4te Mittheilung, 1878.

of the conjunctiva and the reflex closure of the eyelids as distinct as in the normal condition."[1]

Our experiments upon the occipital lobe have yielded no less definite *positive* results than those upon the angular gyrus yielded *negative*.

It may be mentioned in the first place, that we have had no difficulty in obtaining movements of the eyes by applying electrical stimulation to the occipital lobe, and I fail to understand how Dr. Ferrier was unable ever to succeed in getting the same result.[2] Even when no effect can be obtained from the angular gyrus, the surface of the occipital lobe gives a well-marked reaction even to weak excitations. Luciani and Tamburini have also obtained positive results from excitation of the occipital lobe.

In illustration of the effects produced by complete removal of the occipital lobe, and of this alone, I will give two instances,

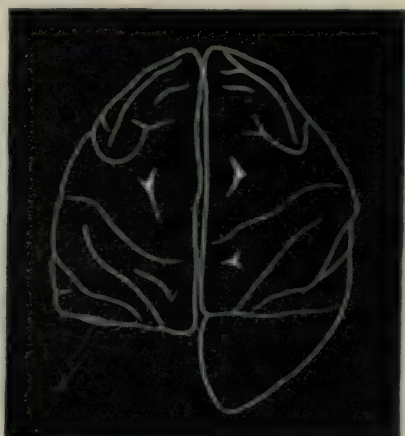


FIG. 2.—Brain of a Monkey, with complete removal of the left occipital lobe. Viewed from above.

in one of which the operation was unilateral, in the other bilateral.

In the monkey upon which the unilateral operation was performed, the left occipital lobe was removed by a vertical incision carried along the line of the parieto-occipital fissure.

[1] 'Functions of the Brain,' 2nd edition, p. 283. [2] Ibid. pp. 244, 271, 288.

That the removal was exact and complete was confirmed on post-mortem examination, some eight months after the establishment of the lesion, when it was seen that the whole of the occipital lobe, and only this lobe, was involved (see Fig. 2), the angular gyrus being quite intact and normal, and the surface of the section looking as fresh, and showing as clearly the distinction of grey and white matter, as if the operation had but just been performed. The result was the immediate establishment of bilateral homonymous hemianopsia, which persisted during the whole time that the monkey was kept alive. Objects so placed that their images fell upon the left half of the retinæ were taken no notice of: a threatened blow coming from the right-hand side of the mesial visual plane was not winced at or avoided; currants strewn upon the floor were only picked up towards the left side, the animal working round in that direction.

In the case of the monkey with a bilateral operation, the lesion was no less complete on both sides (Fig. 3). Here,

a b

FIG. 3.—Brain of a Monkey, with complete removal of both occipital lobes. Viewed from above in *a*, and from below in *b*. On the under surface the lesion of the cortex, as shown by the shading, extends somewhat in advance of the limits of the occipital lobe.

again, the angular gyri were quite intact and perfectly healthy. The result was total and persistent blindness. The animal could only find food by groping and smelling. Brought into

a strange place, it ran against every obstacle. Placed in a dark room and with light flashed upon it, no signs of perception were given. Hearing was very acute, and all the other senses besides vision were unimpaired.

This monkey was in the same physiological condition as one described by Drs. Ferrier and Yeo,[1] and nearly all the description which they give of the behaviour of their animal is applicable to this one. In that case, however, they destroyed both angular gyri by the cautery, besides removing both occipital lobes bodily. It seems clear, however, from our experiment, that the destruction of the angular gyri was unnecessary, since precisely the same results are got when the occipital lobes alone are removed. This result was not, however, got by Drs. Ferrier and Yeo. When one or both occipital lobes were removed by them, they failed to obtain *any symptoms of defective visual perception*. This is, however, contrary to the statements of all other experimentalists, who have invariably obtained hemiopic symptoms transitory or permanent. The explanation may partly be that in Ferrier and Yeo's cases the removal was very incomplete. Thus, in experiment 9, they speak of having severed both occipital lobes with the galvanic cautery and scooped them out bodily. But on referring to the photographic representation of the brain, it is seen that only a small portion of each lobe has actually been removed. And it is the same with other experiments. This also applies, in some measure, to the experiments of Mr. Horsley and myself on the occipital lobes. On completely (as we thought) removing them, we obtained transient hemiopia or blindness, according as one or both sides were operated on. But the post-mortem examination showed that the removal, although more extensive than in Ferrier and Yeo's cases, was really in no instance complete.

Hitherto, Munk is the only observer who has stated that removal of the whole occipital lobe, and of this alone, in monkeys produces immediate and persistent hemiopia, or total blindness,[2] according as one or both sides are operated on. But there was always room to question the exact localisation

[1] *Op. cit.* experiment 12, p. 502.

[2] There seemed to be some doubt as to the persistence of total blindness.

of the lesion in Munk's experiments, because the operations were not antiseptic, and the neighbouring angular gyrus might have become subsequently involved. Moreover, Munk is chary of details, and gives no representations of the brains operated on, but merely a general diagram to illustrate his results.

It would appear, therefore, both from Munk's experiments and our own, that removal of the occipital lobe alone of one side is sufficient to produce permanent hemiopia, and that removal of this lobe on both sides of the brain is productive of complete blindness. But the following experiment shows (1) that, for the production of complete blindness, the removal of the lobe must be complete; and (2) that when a small portion of one of the lobes is left, although blindness is not complete, yet the limit of the visual field of the retinæ may be greatly restricted. An operation was performed with the intention of removing entirely both occipital lobes, and it was done at about the same time, and on a monkey of the same kind and size as the animal whose case we have already related, and in which total blindness was produced. But in the present case the blindness was not quite absolute. From the first the animal appeared to distinguish between light and darkness, and to be conscious of the presence of large objects held between his eyes and the light. Soon (in a day or two) the visual perceptions became better marked, and it was found that, although objects whose images fell upon the upper part of the retinæ remained entirely unnoticed—so that, for instance, food on the floor was not found, and blows aimed from below were not avoided—objects held above the horizontal visual plane were seen with greater or less distinctness, according to their position: those being most clearly seen and most unerringly seized which were held above (and perhaps a little to the right). It appeared, therefore, that this was a case indicating some localisation within the visual area. Munk has in fact described a correlation between the parts of the retina and of the occipital lobe as having been experimentally determined by him in dogs and monkeys.[1] It became, therefore, a matter of considerable interest to examine the brain, and to compare

[1] H. Munk, *op. cit.* 'Fünfte und siebente Mittheilungen.'

it with that of the other monkey with a similar lesion, but with total abolition of visual perceptions. It was found that in the latter case the lesion extended further forwards on the under surface of the brain than in the one in which vision was not totally abolished. The lesion was otherwise equally complete in both cases.

The exact superficial extent of the lesion is shown in Fig. 4.

No other lesions but these total removals of the occipital lobe have produced permanent blindness in our experiments; but we have frequently got temporary hemiopia as a consequence of extensive lesions of the temporal lobe, and also in one case (already related) of the angular gyrus. These

a *b*

FIG. 4.—Brain of a Monkey, with complete removal of both occipital lobes. Viewed from above in *a*, and from below in *b*. It will be noticed that the cortical lesion does not extend so far forwards on the under surface in this brain as in the one shown in Fig. 3.

transient symptoms may perhaps be ascribed rather to the disturbance in the circulation of the occipital lobe, and to the temporary loss of support which is afforded to that lobe by the adjoining parts of the brain, than to the fact, that the visual area of the cortex spreads over from the occipital lobe into the adjoining parts of the brain.[1] If this were the case in the monkey, there should be always some remains of the visual

[1] This is the view adopted by Luciani, "On Sensorial Localisation in the Cortex Cerebri;" 'BRAIN.' Vol. VII. 1884.

perceptive faculty after removal of the occipital lobes alone; whereas both Munk and ourselves have found that there may be no trace left of such faculty. The nearer to the occipital lobe that any such lesion of the temporal lobe is carried, the greater probability is there of the occurrence of (temporary) hemiopia, while extensive lesions of the more anterior parts of the brain fail to yield any sign of visual disturbance. It is, however, also possible that fibres connected with the cortex of the adjoining parts of the brain (and especially of the angular gyrus) may curve backwards into the occipital lobe, and thus become cut off along with that lobe. Various facts might be cited in support of this idea, and it would tend in great measure to reconcile the conflicting statements of experimentors on this region, but we are not yet in a position to come to a definite decision upon the subject.

I have further endeavoured, also in conjunction with Dr. Sanger Brown, to determine whether any evidence is to be obtained from monkeys regarding the localisation of the senses of hearing, smell, and taste. Respecting the sense of hearing, the opinion that has obtained most currency in this country is that of Dr. Ferrier, to the effect that it is entirely localised in the superior temporal gyrus; but, when we come to sift the experimental evidence in favour of this view, we find it to be very insufficient. It is briefly this. (1) that electrical excitation of this gyrus produces movements of the opposite ear (described as "pricking" by Ferrier) and of the head and eyes to the opposite side,[1] (2) that in one monkey destruction of this gyrus upon both sides of the brain has produced total and persistent deafness.[2] I call the evidence insufficient because, in the first place, excitation of very various parts of the cortex (frontal region, middle temporal gyrus, angular gyrus, and occipital lobe) produces almost precisely the same effects, and in the second place, because although it is not difficult to substantiate hearing in monkeys, it is difficult to substantiate deafness, for quite normal monkeys will often fail to pay the least attention even to loud sounds, and in a single case which had been operated on such lack of attention might

[1] Ferrier, 'Functions of the Brain,' 2nd edition, p. 305.

[2] Ferrier and Yeo, *op. cit.*, experiment 13

be erroneously ascribed to loss of hearing, unless it had been ascertained before the operation that the animal invariably reacted to certain noises.

We accordingly determined to repeat Dr. Ferrier's experiments in a number of monkeys. We had no difficulty in obtaining, as a result of electrical excitation of the superior temporal gyrus, the movement of the eyes towards the opposite side, the lids being usually at the same time raised. This effect occurs by excitation along about the posterior or superior two-thirds of the convolution, and it is not confined to this gyrus, but is also obtained on excitation of the adjoining part of the next temporal gyrus.[1] Excitation at the tip of the superior gyrus, just in the angle where the parallel and sylvian fissures meet, produces retraction of the opposite ear. We have not got the pricking forward of the ear which Dr. Ferrier describes.

In six monkeys we have more or less completely destroyed the superior temporal gyrus upon both sides. I say more or less completely, because in one or two a small shred of grey matter belonging to this convolution was found post mortem, but practically the lesion was complete in all six, some of the grey matter within the fissures bounding the gyrus being all that could be taken to represent the convolution, and even this being deprived of its medullary centre. I have no hesitation in affirming that in every case the removal was as complete as—probably more complete than—in the monkey described by Ferrier and Yeo;[2] this I judge from the photographs of the brain and of the sections through it and from the woodcuts which Dr. Ferrier gives in his book,[3] which are clearer than the photographs. But in order to make assurance doubly sure, we in one monkey, a large female Rhœsus, separated up the fissures bounding the gyrus and scooped it out entirely from the very bottom of the fissures, so that not a trace of the convolution in question should remain (Fig. 5). In all six cases the result was the same. Hearing was not only not permanently abolished, it was

[1] Compare Luciani and Tamburini, "Sulle Funzioni del Cervello: seconda communicazione," 1879.

[2] Ferrier and Yeo, *op. cit.*, experiment 13.

[3] 'Functions of the Brain,' 2nd edition, Figs. 97, 98

not perceptibly affected. The animals, even immediately after recovery from the anæsthetic, reacted to slight sounds of an unusual character, such as a smacking of the lips or the rustle of a crumpled newspaper. Some of them were under observation for several months, and there was never any doubt in our minds as to the full possession of their auditory faculties. Nor could the reactions they exhibited to sounds be explained by supposing that they only responded in a reflex manner, for they gave every evidence of understanding

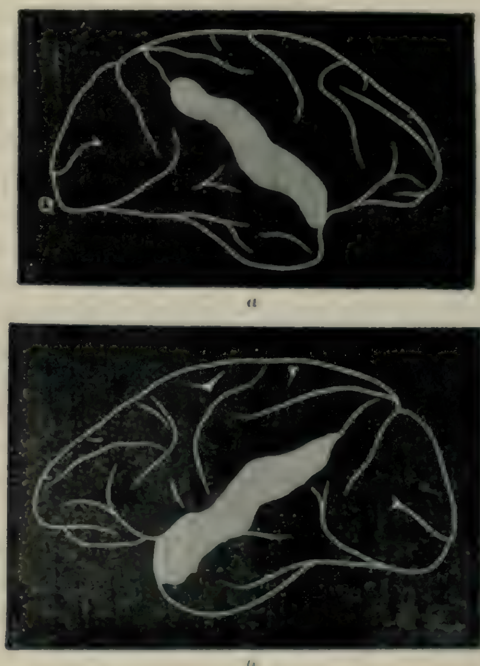


Fig. 5.—Brain of a Monkey, with complete removal of both superior temporal gyri. The right side is shown in *a*, and the left side in *b*.

the nature of different sounds, such as that caused by turning a door-handle or the differences between the footsteps of different people; varying emotions being exhibited according to the anticipations (of food, &c.) which the sounds called forth.

One of the six monkeys had not only the superior temporal

gyri removed, but the whole temporal lobe on both sides, the lesion extending as far as the hippocampal margin of the hemisphere (Fig. 6, *a*, *b*, *c*). This profound double lesion was effected in two operations, and at first produced a condition resembling idiocy, which was well marked for a few days, but afterwards gradually passed off. This condition was probably caused by the great disturbance of the functions of the whole brain which so extensive a removal produced, and especially the vascular disturbance caused by the occlusion of branches of the middle cerebral arteries. A similar condition was brought about in the large female Rhœsus above mentioned, in which both superior temporal gyri were radically removed after separating up the fissures bounding them, and here also there must have been considerable interference with the middle cerebral arteries.

The condition was marked by loss of intelligence and memory, so that the animals, although they received and responded to impressions from all the senses, appeared to understand very imperfectly the meaning of such impressions. This was not confined to any one sense, and was most evident with visual impressions. For even objects most familiar to the animals were carefully examined, felt, smelt and tasted, exactly as a monkey will examine an entirely strange object, but much more slowly and deliberately. And on again, after only a few minutes, coming across the same object, exactly the same process of examination would be renewed, as if no recollection of it remained. The disposition also became completely changed: both animals exhibited the utmost greediness, losing all the daintiness which characterises the feeding of monkeys; they also entirely lost their fear of man. This idiotic condition lasted longer in the large female Rhœsus than in the monkey deprived of the whole of the temporal lobes, which in a few days had already almost entirely recovered memory and intelligence, while the former remained in a stupid condition for some weeks. But there was never any difficulty in observing that sounds, even slight in intensity, were heard. One of these animals was kept for eight, the other for nine months; and except for the condition just described, which

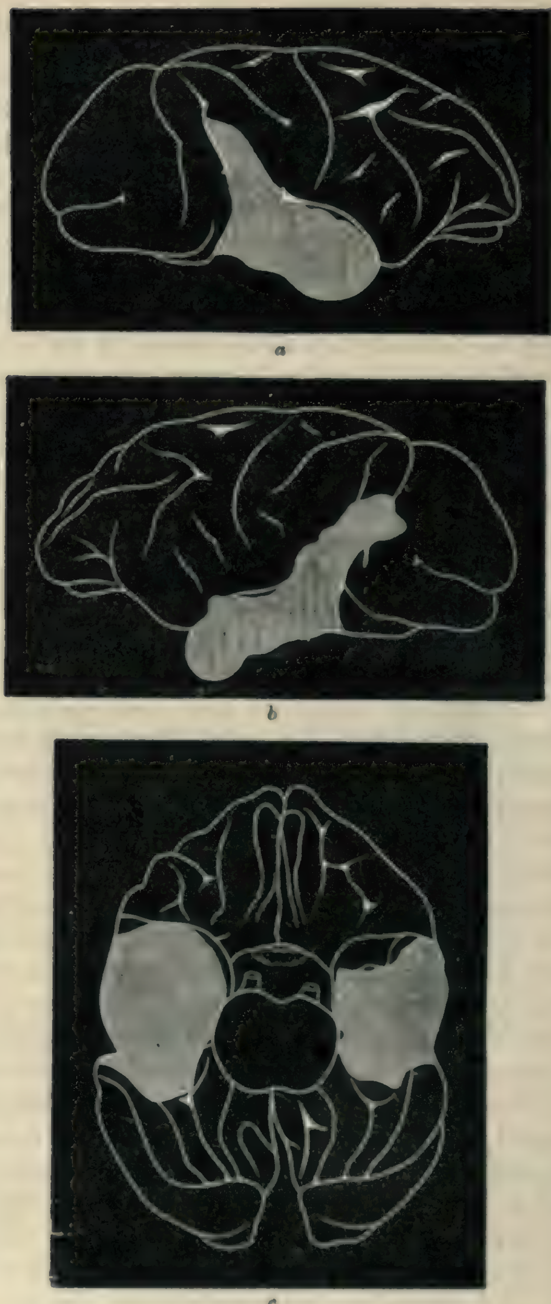


Fig. 6.—Brain of a Monkey, with almost complete removal of both temporal lobes. The brain is shown as seen from the right side in *a*, from the left side in *b*, and from below in *c*.

was only temporary, they behaved during the whole time in every way like normal monkeys.[1]

The case of the monkey with both temporal lobes completely removed militates as strongly against the view, that the auditory perceptive faculty is *localised in those lobes*, as do the six cases against Dr. Ferrier's view. H. Munk appears to be of that opinion; but I cannot find any account of experiments which he has made upon *monkeys* in corroboration of the view; it appears to rest entirely upon experiments on dogs.[2] Luciani and Tamburini[3] speak of the auditory centre in monkeys as being presumably situated in the superior and middle temporal gyri; but this opinion appears to be based merely on the results of electrical excitation. Luciani, in a later communication,[4] remarks, "Our results tend to show that not only the whole cortical area of the temporal lobe, as admitted by Munk, but probably also the *cornu ammonis* forms an integral portion of the auditory sphere." With bilateral extirpation, "at first the auditory troubles may amount to absolute deafness, but soon pass into a condition of obtuseness of hearing. . . ."

Although it is not specially stated, it would appear that these statements (of Luciani) also rest mainly, if not entirely, on experiments on dogs. I believe, therefore, I am justified in asserting, that the supposed localisation of the auditory

[1] These two animals were exhibited to the Neurological Society, and a Committee of that Society was appointed to examine them. Another monkey which was erroneously believed to have been submitted to the same operation (removal of both superior temporal gyri), and a fourth, which had been subjected to no operation at all, were also put before the members of the Committee. No doubt was raised as to the hearing of the first two; but one member of the Committee thought that the third, and another member that the fourth, monkey was totally deaf! This I mention to illustrate the difficulty that even skilled observers, unacquainted with the habits and disposition of the individual animals, experience in deciding the question of the possession or loss of hearing in monkeys. We ourselves had obtained so many evidences from time to time by being frequently with the animals, that we never had the slightest doubt upon the subject; nor could any one that had much to do with them.

[2] *Op. cit.* 4te Mittheilung, Fig. 4, s. 63. In the description of this figure the author states, "Mit *B* ist die Rindenpartie bezeichnet, welche nach den Erfahrungen am Hunde als Hörsphäre anzunehmen ist." Munk regards the supero-posterior tip of the first temporal gyrus and the adjacent part of the angular gyrus as the "sensory area of the region of the external ear."

[3] *Op. cit.*

[4] "On Sensorial Localizations," etc., 'BRAIN,' Vol. VII. p. 154.

perceptive faculties in the temporal lobe in monkeys has no experimental evidence in its favour, and the case I have described, in which both temporal lobes (including the *cornu ammonis*) were wholly removed without any permanent diminution in the acuteness of hearing, bears strongly against that view.

In the same animal smell and taste were also present, and, so far as we could ascertain, were in no way diminished in acuteness. This was also, as may *à fortiori* be expected, the case in two other monkeys, in which we cut away the antero-inferior extremity of the temporal lobe on both sides. These experiments were made previous to those of total extirpation of the lobe, and were intended to test Dr. Ferrier's surmise, that this region of the temporal lobe is related to the faculty of taste.[1] But they give no support to that surmise (which is besides based on entirely insufficient evidence[2]), for all our animals gave well-marked evidences of taste both in the selection of favourite articles of food and in the disgust exhibited when a bitter substance, such as quinine, had been concealed within a raisin and thus inadvertently taken into the mouth.

With reference to the cerebral localisation of perceptions produced through the nerves of tactile and of general sensibility, I would recall the fact that, in the researches which I have before alluded to as having been carried on in conjunction with Mr. Horsley,[3] we described a number of experiments upon monkeys in which a more or less extensive destruction of one gyrus fornicatus was produced; this lesion being invariably accompanied by a considerable diminution, or even by complete abolition, of tactile sensibility on the opposite side of the body, while at the same time there was also considerable loss of general sensibility. This condition was always followed

[1] 'Functions of the Brain,' 2nd edition, p. 321.

[2] Movements of the lips, tongue, and cheek pouches on electrical excitation (which may well have spread to the superjacent motor area in the lower part of the frontal lobe) and certain earlier experiments in which lesions (not aseptic) were roughly produced in this part, and loss of taste seemed to follow. But the animals lived but a short time, and were in an entirely abnormal condition when tested, nor was the testing performed with purely sapid substances, but with acid and aromatic articles.

[3] 'Philosophical Transactions,' 1887.

by partial recovery, which gradually supervened until a stationary condition of diminished sensibility appeared to be permanently established. We did not, however, keep any of our animals which had been subjected to this operation for more than about three months. In order, therefore, to determine if possible whether in such cases there was ultimate complete recovery, I removed in another monkey a portion of about one and a half centimetres in length (the whole brain being about six centimetres long) from the middle of the gyrus fornicatus, on the right side of the brain. This operation produced a condition of almost complete (hemi-) anæsthesia, involving the whole of the left side of the body with the exception of the forearm and hand. There was a little paresis of the leg, due apparently to a portion of the leg area in the superjacent marginal convolution having been accidentally injured in the operation, but none of the upper limb or of any other part of the body. As a rule, there was no reaction to touch, nor even to a slight prick applied to the anæsthetic side, and even when a movement followed, it was more like a reflex action than one dictated by the higher centres, for it was unaccompanied by wincing, which was invariably produced when the other side of the body was suddenly touched or pricked.

This animal was kept under observation for more than seven months, and was tested from time to time. It was found that although the condition in question underwent some improvement, there was still during the whole of that time a marked difference in the two sides of the body, the left side, with the exception of the forearm and hand, which were never anæsthetic, being distinctly deficient in sensibility as compared with the right. We may fairly infer, therefore, that the hemianæsthesia which is produced by destructive injuries of the gyrus fornicatus is in all probability permanent. Whether however the perception of impressions of tactile and general sensibility is confined to the gyrus fornicatus and its continuation into the gyrus hippocampus, can only be effectually cleared up by the entire removal of these parts of the limbic lobe—an operation of the greatest difficulty, but one which I

have not given up hopes of ultimately effecting. In the meantime, I would affirm the extreme probability of this hypothesis on evidence obtained by exclusion, for I have never been able to determine the existence of any permanent diminution of sensibility after operations upon other parts of the cortex, unless there were a possibility of the limbic lobe having been directly or indirectly involved.

ON INJURIES OF THE CAUDA EQUINA.

BY WILLIAM THORBURN, M.D., B.S., B.SC. (LOND.), F.R.C.S.

Surgical Registrar to the Manchester Royal Infirmary, &c.

THE series of cases upon which the conclusions drawn in the present paper are based, appear to the writer to form a clinical picture the import of which has not hitherto been fully recognised, although its features are sufficiently marked and the symptoms described have been noted by more than one observer. They present instances of very different lesions, all of which, however, agree in the production of pressure on the cauda equina, and although they are not all injuries, it has appeared advisable to place side by side with traumatic cases, those due to other causes, but resembling them in the locality of the lesion and the nature of the symptoms. All these cases have been treated in the Manchester Royal Infirmary, to the courtesy of whose physicians and surgeons I have again to express my indebtedness for permission to use the material under their care.

I shall in considering these cases first relate their clinical histories, drawing attention to the salient points of each, and shall then proceed to draw certain conclusions, and to compare the facts observed with the experience of previous writers.

CASE I.—*Dislocation of the First Lumbar Vertebra.—Compression of the Cauda Equina.*

P. S. attended as an out-patient in Dr. Ross's clinic during the month of June 1886, and was admitted to the wards on the 7th of the following month. He gave a history of having been a heavy smoker and drinker, of an attack of syphilis twelve years ago, and of pneumonia five years ago. On Jan. 31st, 1886, he fell from a scaffolding and injured his back. For five days he was un-

conscious, and has since then had paralysis of the lower limbs with retention of urine, requiring the constant use of a catheter.

On examination, he presented a distinct deformity of the lumbar spine, there being a wide interval between the first and second spinous processes with prominence of the latter. There was slight pain in the affected region, but no tenderness. (The exact position of the deformity was verified by repeated examinations, by various gentlemen.)

The lower limbs presented complete paralysis of all the muscles below the knee, and of the flexors of the knee, and there was weakness but not entire loss of power in the extensors of that joint. Flexion of the thigh could apparently be performed without difficulty; the power of adduction was slight, and that of abduction almost, but not quite entirely lost. The buttocks and lower extremities were wasted throughout. Electric examination of the affected region gave contractions with the following currents:—

	Right Limb.			Left Limb.		
	Cathodal Closure.	Anodal Closure.	Faradic Current.	Cathodal Closure.	Anodal Closure.	Faradic Current.
	cells.	cells.		cells.	cells.	
Rectus	..	..	No effect	..	..	No effect
Sartorius	35	30	,,	25	20	,,
Vastus Externus . .	25	20	,,	25	30	..
Vastus Internus . .	35	30	,,	30	30	..
Gluteus maximus . .	No contraction with 50 cells.		,,	No contraction with 50 cells.		..
Biceps			,,			..
Semimembranosus .			,,			..
Semitendinosus . .			,,			..
Adductor Longus . .	..	..	,,	..	..	..
Adductor Magnus. .	..	..	,,	..	..	..
Gracilis	..	..	,,	..	..	..
Gastrocnemius. . .	40	40	,,	35	30	..
Tibialis Anticus . .	25	20	,,	25	20	..
Extensor Proprius Pollicis	..	..	,,	..	..	..
Peroneus Longus . .	25	25	,,	30	30	..

The knee-jerk and plantar reflexes were absent; but the cremasteric reflexes were normal.

The urine was retained, and had to be drawn off by the catheter; fæces were passed involuntarily.

Sensation was normal on the upper part of the buttocks, that is, in the region supplied by the last dorsal, ileo-hypogastric and external cutaneous nerves, and was little if at all diminished on the front of the thighs and the anterior halves of their inner and

outer aspects, or on the inner sides of the legs; but there was complete anæsthesia of the backs of the thighs, of that part of the buttocks not included in the above limits, of the outer sides of the legs and of the feet. The perineum, the penis, and the scrotum were also quite anæsthetic, with the exceptions of the root of the latter, and the catheter was not felt in the urethra. The patient was, however, aware when the bladder was full, and when he wished to empty the rectum, but had no control over the latter, and could not feel the passage of fæces. At times he would have pricking sensations in the toes and some pain in the thighs.

The lower limbs presented no obvious change of temperature. Since the accident he had had no erections of the penis. On both heels were bedsores of large size. The urine was alkaline, containing large quantities of pus and phosphates.

The patient remained under observation and treatment for some time; but, with the exception of amelioration of his cystitis and bedsores, underwent no change. On leaving, he was instructed to return, with a view to trephining the spine, but has not since been heard of.

The explanation of the above case is sufficiently obvious. There is sensory paralysis of all the nerves of the sacral plexus and possibly of the obturator, but not of the anterior crural or other lumbar nerves: the perineum, penis, scrotum and urethra, being supplied by branches of the pudic, are anæsthetic, but the root of the scrotum retains sensation owing to the presence of twigs of the ileo-inguinal nerve, which, however, only descends to a very short distance.

As regards motion, we find complete paralysis with the "reaction of degeneration" of the muscles supplied by the nerves of the sacral plexus. Those supplied by the anterior crural, although presenting the reaction of degeneration, are only weakened, and the adductors, supplied by the obturator, appear also to retain some power.

Again, the cremasteric reflex remains, but below this point reflex action is lost.

Case II.—*Spina Bifida.—Cure.—Cauda Equina in Cicatrix.*

F. H. W. has been several times admitted into the Manchester Royal Infirmary, under the care, successively, of Mr. Lund, Mr. Whitehead, and Dr. Ross. He is a clerk by occupation, is 24

years of age, and gives the following account of himself. At birth he had a swelling (spina bifida) of the lower part of the back, which was never larger than an orange. Very soon after birth a needle was thrust into this, but he does not know whether any effect resulted. When two years of age he was said to have had a fit, followed by paralysis, and subsequently wasting of the muscles below the knee on both sides. He also states that there was some contraction of the calf muscles, causing drawing up of the heel, which on two occasions required division of the tendo Achillis, followed by the use of a metal boot. The deformity was thus eventually overcome. When about fifteen years of age he began to be troubled by an ulcer on the outer side of the right foot, which resisted all treatment, until in 1883 the little toe with its metatarsal bone was amputated by Mr. Lund. The wound thus caused remained open for nineteen months, at the end of which time its upper end had again formed an ulcer. This ulcer still remains, and is his chief trouble; it improves when he is confined to his bed, but soon breaks down again when he tries to move about.

The condition of the patient never varied very materially at the various times, extending over a period of some eighteen months, during which he was under observation, and he presents the following points.

On the back, opposite to the last lumbar or first sacral vertebra, is a flattened swelling about the size of a hen's egg, but of lenticular shape and covered with hair. At its centre is a depression, into which he states that a stocking-needle was passed at birth; but he also says that the depression was congenital, and that the needle was used only to probe its depth. The swelling is of an elastic consistence, and gentle manipulation caused sensations which the patient says are pleasurable but indescribable. Firm pressure causes passage of urine, defecation and strong sexual desire: a blow upon it causes some rigidity of the legs. Over the swelling is a luxuriant growth of hair, which is also well-developed on the lower limbs.

Both the lower limbs show distinct wasting, which is more marked on the right than on the left side, the circumferences being: right calf, 8 inches; left calf, 11 inches; right thigh, 14½ inches; left thigh, 17 inches; while the right is half an inch shorter than the left limb. On the right side, the fifth toe and its metatarsal bone were removed, and on the outer side of the foot over the fourth metatarsal is an oval ulcer about one inch long and half an inch wide. The ulcer shows a clean-cut margin, which is raised, horny and thickened, with slight undermining of the edges and

pale granulations at the base. Between the second and third toe on the same foot was a second small ulcer which recovered with rest. The arch of the foot is exaggerated, the toe pointed, and there is no power of movement about the ankle-joint.

The lower limbs are partially paralysed, with weakness and wasting of most of the muscles, and especially of those below the knee, the leg muscles of the right side being completely paralysed. The knee-jerk, ankle-clonus, and the plantar reflex are absent on both sides; the cremasteric, abdominal, and epigastric reflexes normal. The electric reactions of the muscles were as follows:—

	Right Side.		Left Side.	
	K. C. C.	A. C. C.	K. C. C.	A. C. C.
	cells.	cells.	cells.	cells.
Sartorius	40	40	35	40
Adductor Magnus	25	30	35	45
Gluteus Maximus	nil	nil	50	nil
Vastus Externus	25	40	40	50
Vastus Internus	45	40	45	nil
Gastrocnemius	45	50	25	40
Peroneus Longus	50	nil	40	35
Tibialis Anticus	45	45	40	nil

Hence they did not present the "reaction of degeneration." To the faradic current they reacted with difficulty on both sides, the anterior muscles of the left thigh acting most readily, those of the right foot not at all. He has difficulty in walking, being always afraid of falling, and in the dark he staggers and has to grope his way. The walk is characteristically "pseudo-tabetic," the toe dropping at each step. At times, especially if he is tired, there are slow fibrillar movements of the muscles of the right thigh and gluteal region, with occasional choreiform movements of the right foot.

As regards sensation, he states that he cannot judge of the position of his right lower limb, and that the ground does not feel solid under his feet. At times the limbs feel "as if they did not belong to him, but were some distance off." He occasionally has pain in the dorsum of the right foot, the knees and hips, and intense tickling sensation in the sole of the right foot. On examination, there was found to be extensive anæsthesia of the lower limbs, of similar distribution on both sides. The affected area was not quite sharply defined, but had the following general outline as represented in the accompanying diagrams, where the

anæsthetic portion is shaded. Commencing above at the side of the tumour and almost at its centre, the boundary-line runs downwards and outwards, across the upper limit of the gluteal region, thence over the great trochanter down the outer side of the thigh to the apex of the line leading to the external condyle; it now tends forward, somewhat to the front of the condyle, and then down along the line of the fibula for about half its length; after which it comes forward and inward across the shin, ending about the middle

Fig. 1.

Fig. 2

of the first metatarsal bone. The inner boundary commences about the external inguinal ring, passes outward towards Poupart's ligament; thence slightly backwards for a short distance; again down the inner aspect of the thigh to the back of the internal condyle; thence down the inner side of the leg, curving below the internal malleolus and running forwards to join the former line over the metatarsal bone of the great toe. It will be noted that these limits include the gluteal region, back of the thigh, the back

and partially the outer side of the leg, and the whole of the foot, except a small area on its inner aspect. Further, the perineum is included in these boundaries, being absolutely anæsthetic. The penis also is anæsthetic, except at its extreme root, as is the scrotum, except along a line, too small to represent in the diagram, extending forwards and downwards from the external ring for about two inches, and corresponding apparently to the distribution of the ileo-inguinal nerve. Although the scrotum is thus anæsthetic, testicular sensation on deep pressure is normal.

At one period he states that he passed urine involuntarily, and had to wear a bag to catch it; but he can now retain it, and indeed only passes it with difficulty and much straining. He knows when the bladder is full. The bowels are usually relaxed, and at times he has involuntary evacuations. He is not always able to tell whether he has or has not passed urine or fæces. He has sexual sensations and enjoyment, but states that on connection the semen is usually ejaculated before intromission, but that on a second coitus he can perform the act as usual. When under the influence of drink he says that he can both pass his urine without difficulty and complete the sexual act on the first attempt.

He is subject to attacks of lymphangitis and swelling of the inguinal glands in the right lower limb, which attacks he believes to have a tendency to monthly periodicity, and to be brought on in many instances by drinking or by sexual excitement.

This case resembles the first very closely, differing mainly in the less complete paralysis and in the partial reaction of degeneration replacing the complete degeneration shown by Case I. The distribution of the anæsthesia is similar to that of Case I. An interesting point is the retention of sexual desire and enjoyment in spite of the complete anæsthesia of the penis, and the evidence of persistence of sensation in the testicles which derive their sensory nerves from some point higher in the cord than does the scrotum. The paralysis is again seen to affect mainly the branches of the sciatic, gluteal, and pudic nerves, sparing the anterior crural and obturator with the upper lumbar branches. That the lesion is an involvement of the cauda equina in the cicatrix of the spina bifida there can, I think, be no doubt. The trophic lesion of the right foot is interesting, and is similar to that seen in a case reported by Ogston[1] of old spina bifida with perforating ulcer of the left foot, anæsthesia

[1] 'Lancet,' 1876, vol. ii. p. 13.

of the outer side of the leg, and dorsal and plantar aspects of the foot and diminished faradic contractility of the muscles of the foot, all on the same side. Indeed Ogston's case is clearly of the same nature as the above, differing only in that the cicatrix had, in his case, involved but a portion of the fibres of the sciatic of one side only.

Another case of the same nature is reported by Brunner[1] as an instance of *spina bifida occulta*. The patient had a depression over the spine from the first to the fifth lumbar vertebra, excessive growth of hair over that region, and a perforating ulcer on the outer side of the right foot. The right lower limb was wasted, especially below the knee, and there was some loss of power in it: there was anæsthesia of the sole and outer side of the foot; the knee-jerk was lost.

The next case which I shall quote presents similar symptoms arising from the pressure of a tumour on the cauda equina.

Case III.—*Tumour of Cauda Equina.*

Joseph Davies was admitted under the care of Dr. Ross on May 12th, 1882. His previous history presented nothing of interest. About five months before admission he began to suffer from pains shooting from the small of the back down the backs of the thighs and legs to the feet, which gradually increased until he was unable to bend his back and could hardly walk.

On admission he complained of the above pain, and of great pain in the buttocks when sitting down. He could hardly walk, dragging the legs along the ground slowly and with difficulty, and the lower limbs were much wasted. The patellar reaction was increased on both sides. The urine was retained, and had to be drawn off with a catheter. Pupils presented no abnormality. He was treated with strychnia and iron.

The notes at this period are very imperfect, but there seems to have been little or no change for a long time. On July 5th he was ordered gr. 5 doses of pot. iod. On July 13th it was noted that pain was greatest about the ankles and outer sides of the feet. There was no staggering in the gait, nor did he sway when standing with the eyes closed, but the movements of the lower limbs were very feeble, especially those of the gluteal muscles. The plantar and cremasteric reflexes were well marked, but the gluteal was sluggish. The patellar tendon reaction was lively (? exag-

[1] Virchow's 'Archiv,' 1887, p. 494. See Appendix.

gerated) on both sides. On both sides the muscles of the lower limbs were markedly atrophied, and with the exception of the gluteus maximus of the left side, had lost their faradic contractility, this muscle also only reacting to strong currents. Analgesia and diminution of tactile sensibility was present over the back of the sacrum, extending thence to the perineum, the left side of the scrotum, the backs of both thighs, and down the calves in the form of a triangle with the apex downwards.

Four days later the anæsthetic area was found to have extended, so as to involve the buttocks up to the level of distribution of the ileo-hypogastric nerves, the backs of the thighs, and the bulk of the leg, omitting, however, the knee-joint, inner border of the tibia and foot, and the great toe (i.e. the distribution of the internal saphenous nerve). The anæsthesia was less perfect in the legs than in the thighs and buttocks. His urine was still retained, but he drew it off himself with a catheter. On going to stool, he could not pass a motion until he had pressed upon the perineum, but very light pressure—even merely wiping the anus—was sufficient, so that the action was probably not entirely mechanical. Pain in the lower limbs was very severe, especially on movement, and he had often much pain about the anus; those symptoms being so severe as to necessitate hypodermic injections of morphia.

A month later he could still move the lower limbs in all directions, but only with the greatest difficulty, and apparently somewhat better on the right than on the left side; there was also extreme wasting of the limbs, but it was difficult to say that one group of muscles was more affected than another. The plantar and cremasteric reflexes were exaggerated, the gluteal absent on both sides. The knee-jerk was, as before, well marked, but there was no ankle clonus. With the faradic current the gastrocnemius and glutei gave no reaction; the anterior leg muscles and all those of the thigh reacted to a current of medium strength. The following table shows the number of cells required to produce contraction with the constant current:—

	Cathodal Closure.	Anodal Closure.
	cells.	cells.
Biceps (right)	30	30
,, (left)	35	30
Gluteus maximus (right) . . .	20	20
,, ,, (left)	15	25
Extensors of foot (right) . . .	40	40
,, ,, (left)	40	40
Extensors of knee (left) . . .	30	45
Gastrocnemius (right)	30	35
,, (left)	45	45

FIG. 3.

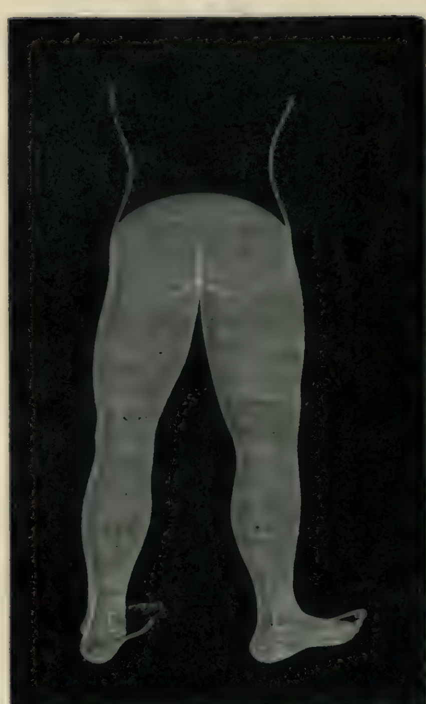


FIG. 4

FIG. 5.

FIG. 6.

The distribution of the anæsthesia at this date is indicated by the accompanying diagrams (Figs. 3–7). Pain was still very great, but the general health remained fairly good. There was some redness over the trochanters and sacrum.

From this time the patient lost ground rapidly. The skin was sore at several points, an abscess formed over the right trochanter major, the pain was intense, the appetite failed, and the temperature became hectic, varying from 97° in the morning to 103°·6 in

FIG. 7.

the evening. On Sept. 11th he was ordered drachm doses of liq. hyd. perchlor. with grs. ij of pot. iod. three times a day, but no improvement followed. Morphia had to be used constantly.

On Nov. 1st a bedsore formed over the sacrum, and on Nov. 15th he had convulsions and died in the afternoon.

No notes were taken of the post-mortem examination, but Dr. Ross, who was present, and Prof. Young who was at that time Pathologist to the Infirmary, remember that there was found only a very small tumour—a fibro-sarcoma about the size of a hemp seed —situated on one of the nerve cords of the cauda, with no signs of diffused infiltration or inflammation. It is, however, obvious that there must have been some lesion of more than the one nerve root, and no microscopic examination was made of the others. The spinal cord itself was perfectly normal.

We have here the same distribution of anæsthesia as in Cases I. and II., but the notes contain no reference to the relative power of the thigh muscles. The reaction of degeneration was again absent. Owing to the nature of the lesion the anæsthesia was preceded by intense pain, and the symptoms were at first more marked on the left than on the right

side. The retention of the knee-jerk and plantar reflexes is remarkable, but some exaggeration of reflex action is by no means rare in the earlier stages of peripheral nerve lesions, before irritation has given rise to complete annihilation of function. The nature of the lesion was here placed beyond any possible doubt by the post-mortem examination, which showed that the cauda equina and not the spinal cord itself was the region involved.

A closely similar case is mentioned and figured by Gowers,[1] who notes that tactile sensibility was impaired "chiefly in the region supplied from the sacral plexus;" and that although the lower limbs were paralysed, "a little power in the flexors of the hips and extensors of the knees persisted almost to the last." Hence, in Gowers' case also, the anterior crural nerve was less profoundly affected than the branches below it. His figure shows a tumour of the cauda equina immediately below the termination of the cord.

Case IV.—*Dislocation Forwards of the Second Lumbar Vertebra. —Compression of the Cauda Equina.*

R. M. C., aged 15, male, a collier by occupation, was admitted to the Infirmary under Mr. Jones's care, on December 31st, 1886.

About the end of the previous August, while he was in the pit, and probably in a stooping posture, a stone weighing 5 or 6 cwts. fell from the roof—a distance of about 5 ft.—on to his shoulders, bending him forwards with his head between his knees, and his right leg under him. On being extracted, he was found to have a fracture of the right femur, and this was apparently the only injury diagnosed at that time; but he had much pain in the lower part of the back, and in both hips, and was unable to sit up in bed. Some nine weeks later, he could sit in a chair. He was never able to move his feet after the accident, and not for a fortnight had he any power over either thigh. He had never any pain or other unusual sensations in the lower limbs. For six weeks after the accident his urine had to be drawn off systematically with a catheter, and from about the third week he had symptoms of cystitis, which still continued on admission. Since the sixth week no catheter has been used, and he has been able to pass water, nor has he ever had any incontinence; but micturition is very slowly

[1] 'Diseases of the Nervous System,' vol. i. p. 420.

performed. Constipation has been present throughout, but there has never been any involuntary defecation.

On admission we found a prominence of one of the lumbar spinous processes (the third) one inch above the level of the posterior superior iliac spines. Above and below this were depressions, and to the left of, and a little above it, another bony prominence, due apparently to the displaced transverse process of the second lumbar vertebra. There was slight pain and tenderness in this region.

The lower limbs presented partial paralysis. The hips could be moved in every direction, but adduction was more powerful than any other movement; extension and flexion were about equal, and abduction very weak; at the knee extension was more powerful than flexion. In the ankle and foot no movements could be produced. The muscles of the buttocks and lower limbs were wasted, especially below the knees; none of them contracted with a faradic current of such strength as the patient could bear, and we were unable to test satisfactorily the galvanic reactions. The knee-jerk was absent, as was the plantar reflex, the cremasteric and gluteal being well marked and apparently exaggerated.

There was nowhere absolute anæsthesia, but sensation was obtuse over a region fairly well defined by the following boundaries:—above the upper part of the gluteal region, thence down the back of the thigh, leg and sole of the foot, the front of the leg, and the dorsum of the foot. It was much less imperfect on the front of the thigh than elsewhere in the lower limbs, and was better on the inner than on the outer side of the leg. Over the genitals also sensation was much blunted but not absent, and a catheter was felt along the whole of the urethra. The passage of fæces was also felt. He states that sensation had gradually improved since the accident. The feet always felt cold, but there was no pain or hyperæsthesia.

The skin of the lower limbs presented no abnormalities. Priapism was common; the urine was alkaline, containing some pus and phosphates, and he had pain over the pubes, and smarting on micturition.

On January 15th—a fortnight after his admission—Mr. Jones proceeded to "trephine" the spine in the affected region. Chloroform having been administered, the patient was turned on his face, and an incision four inches in length was made in the middle line, with its centre over the prominent spine. From either end of this an incision of some three inches was carried at right angles to it, and to the left. The superficial structures were thus dissected up in a flap, and the muscles were drawn aside from

the vertebral groove, and held back by retractors. It was now clear that the arch of the second lumbar vertebra was displaced forwards, the prominent spine being that of the third. Thus the displacement was that most commonly met with—dislocation forwards of the upper part of the spine; at the same time the spine of the second lumbar was broken off and isolated, and the prominence above mentioned as lying to the left of the middle line proved to be the articular process of the third lumbar, its articular surface being exposed by the dislocation forwards of that of the second. The detached spine of the second vertebra was removed, and showed a gap between the arches of the second and third, filled with dense cicatricial tissue. By means of bone forceps, the arch of the second lumbar was now almost entirely removed, exposing the membranes of the cord which had obviously been compressed by it. Around these membranes there was also cicatricial tissue, which was not interfered with. The flap was replaced and sutured, a drainage tube being placed at its lower angle, and the wound dressed with wood wool.

No trouble followed the operation, and the wound healed well, but rather slowly, the temperature being more or less raised for about a fortnight afterwards. Five days later the patient stated that the sensation of coldness in the feet had disappeared. After a week the faradic current was used to the muscles, and caused slight contractions in the posterior thigh muscles, a more marked effect in the anterior muscles and adductors, but none in the legs. The galvanic current was never used, as the patient could not bear it, and struggled when it was tried. Sensation improved somewhat, and the thigh muscles became much stronger during the ensuing two months, but no power of motion returned in the leg muscles. In this condition he was sent to the convalescent hospital at Cheadle, on April 2nd, two months and a-half after the operation.

A month later, when I saw him at Cheadle, he could stand up, and could, by means of chairs, &c., walk a little; the thigh muscles were fairly developed, and moveable in all directions, but the leg muscles remained atrophied, and he could not move the ankles or toes. The thigh muscles reacted to the faradic current, but those of the legs did not; it was not possible to obtain accurate galvanic observations, but apparently the ascending and descending currents were equally effective in producing contractions of the thigh muscles, and equally unable to affect those below the knees. Occasionally he had muscular tremors. Sensation appeared to be everywhere normal, but was perhaps a little more acute on the inner than on the outer side of each leg. The superficial reflexes

were well marked, the knee-jerk absent. A small quantity of pus still remained in the urine, but there were no subjective symptoms connected therewith. A fortnight afterwards, when I saw him again, he could walk with the aid of one stick only, but with marked dragging of the toes. There was no other change.

The relationship of this case to the three first cited is obvious, and the localisation of the lesion indubitable. The most interesting point in the symptoms is the slight interference with sensation as compared with motion.

We are now in a position to compare the above four cases, which, although differing somewhat in their details, resemble one another sufficiently in their broad outlines to form a distinctly marked group. For this purpose, I have arranged the leading symptoms in the form of a table, showing the similarities and points of difference in each case (p. 396).

It is to be remembered that the spinal cord terminates at the level of the lower border of the first lumbar vertebra, whence the nerves of the lumbar and sacral plexuses descend to pass out from the spinal canal, each immediately beneath the vertebra from which it takes its name. Hence, in all of these cases the lesion was so situated as to compress the cauda equina and the cauda equina only; and further, in Case I., the whole of the lumbar and sacral nerves pass out beneath its level, while in Case IV. it involves all but the ileo-inguinal and ileo-hypogastric; in Cases II. and III. the exact level of the pressure cannot be ascertained, but must clearly have been almost the same as in Cases I. and IV.

In all of these cases the salient point is that, although the whole or almost the whole of the nerves of the cauda pass the level of the lesion, those which escape from the spinal canal lower down are more seriously injured by the pressure. Why this should be so is not clear. Those nerves which come out lower down are, in the cauda, situated nearer the middle line than those which pass out above them, and hence they would appear to have more room to escape from pressure, and we might expect them to suffer less rather than more; but that the contrary is the case is an established fact, and we are able definitely to conclude that, *in a pressure lesion of the entire cauda*

	Case I.	Case II.	Case III.	Case IV.
Lesion	Dislocation of 1st lumbar vertebra.	Spina bifida	Tumour	Dislocation of 2nd lumbar vertebra.
Paralysis . . .	Complete in distribution of sciatic and pudic nerves. Partial in anterior crural and obturator.	Complete in distribution of tibial branches of sciatic. Partial in anterior crural, obturator, sciatic (and pudic).	Partial in all muscles of lower limbs.	Complete in tibial branches; almost complete in upper branches of sciatic. Well marked in anterior crural; less marked in obturator.
Reaction of degeneration.	In distribution of anterior crural, obturator, sciatic (and pudic) nerves.	Partial in distribution of anterior crural, obturator, sciatic (and pudic).	Absent	Doubtful.
Reflexes . . .	Patellar and plantar lost. Cremasteric normal.	Patellar and plantar lost. Cremasteric normal.	Patellar, plantar, and cremasteric marked. Gluteal absent.	Patellar and plantar absent. Gluteal and cremasteric present.
Anæsthesia . .	In distribution of superior gluteal, sciatic, and pudic nerves, and in posterior sacral branches. Slight if at all in external cutaneous, anterior crural, or obturator.	In superior gluteal, sciatic, pudic, and posterior sacral.	In superior gluteal, sciatic, pudic, and posterior sacral.	Partial in distribution of superior gluteal, sciatic, and pudic.
Bladder and rectum	Retention of urine. Incontinence of fæces.	Partial retention of urine, preceded by incontinence. Partial incontinence of fæces.	Retention of urine. Retention of fæces.	Retention of urine. Constipation.
Vaso-motor and trophic.	No erections. Temp. normal. Bedsores on heels.	Erections normal. Temp. normal. Perforating ulcer.	Bedsores at various points.	None.

equina, those nerve roots which emerge lower down are more seriously injured than those above them.

As already stated, the above-described combination of symptoms has been several times noted by former writers, but as in other cases of spinal injuries, the descriptions given are usually so meagre as to render the diagnosis anything but clear. The annexed table (pp. 398–403) gives a few instances, from several hundreds of cases of spinal injury which I have abstracted, showing more or less clearly the same group of symptoms, and indicating the various interpretations that have been placed upon it. The table does not pretend to be exhaustive, but merely illustrative.

I shall not add anything with regard to the spinal symptoms in the above, which are sufficiently similar to my own cases, but merely wish to adduce evidence in favour of their being due to the pressure on the cauda equina. Case 1 is attributed by the author to "intra-spinal hæmorrhage," but the date of appearance and the seat of tenderness appear to me to warrant the interpretation given in the table. Case 2 appears to me to be most typical, and I entirely fail to see why it should be regarded as an instance of "concussion:" the lesion was clearly below the region of the cord itself and over the cauda equina, and the symptoms are those of pressure upon or partial destruction of the latter. In Case 5 Mr. Hutchinson places the probable seat of the lesion at the fourth or fifth lumbar vertebra, a conclusion which, in view of our cases, is hardly warranted; at first all the muscles of the lower limbs seem to have been paralysed (unfortunately, sensation was not at that time accurately noted), thus indicating, I believe, an affection of the entire cauda.

The best description is that of Leyden (Case 8), but he appears to have attributed the symptoms mainly to myelitis, and not to the crush of the cauda equina. In the other cases, the localisation is as a rule clear enough, from the facts above furnished.

An important case is given by Kirchoff,[1] in which there was traumatic dislocation backwards of the first lumbar vertebra; causing paralysis of the lower limbs, retention of

[1] 'Archiv f. Psychiatrie,' Bd. xv.

No.	Reference.	Sex.	Age.	Result.	Probable Lesion.	Paralysis, &c.	Reflexes.
1.	Erichsen, 'Concussion of the Spine,' 2nd ed. p. 30.	M.	14	Recovery in four months.	Inflammation around cauda equina, more marked on left side.	Came on gradually during ten days. Could then not stand, but moved legs in bed; no complete paralysis except in peronei, and extensors of left ankle. Rapid wasting of left leg.	No note.
2.	Lidell, Ashurst's 'System of Surgery,' vol. vi. p. 789.	M.	19	Unknown.	Gunshot wound in lumbar region.	Paralysis below the seat of injury.	No note.
3	Ollivier, 'Traité des Maladies de la Moelle Épinière,' vol. i. p. 358.	M.	40	Partial recovery.	Gunshot wound in lumbar region.	None.	No note.
4.	Hutchinson, 'Lond. Hosp. Rep.' vol. iii. p. 348.	M.	42	Death in five weeks.	Dislocation of 2nd lumbar vertebra.	Lower limbs were paralysed with the exception of the anterior thigh muscles.	No note.
5.	Hutchinson, 'Lond. Hosp. Rep.' vol. iii. p. 326.	M.	?	Recovery in four months.	Upper lumbar region.	Paralysis of the legs. On eighth day could draw up both legs, the right with greater difficulty than the left.	No note.

Anæsthesia.	Bladder and rectum.	Vaso-motor and Trophic.	Local Symptoms.	Post-mortem.	Remarks.
Numbness and tingling on outer side of left thigh; partial loss of sensation below left knee. Right limb normal.	Occasional loss of control over sphincters.	Coldness of extremities, especially of left foot.	Tenderness over 3rd lumbar vertebra after 10th day.	..	There were also symptoms of cervical injury.
Diminished sensation below seat of injury. Hyperæsthesia of front and inner side of thigh. Anæsthesia of ureter anterior to pars prostatica.	Retention of urine.	No note.	Gunshot wound.	..	Author regards as a case of concussion, because the retention of some sensation shows that there was no serious cord lesion (*sic*).
Absolute anæsthesia of postero-internal and anterior parts of thighs; of penis and scrotum.	Incontinence of urine.	None.	Gunshot wound.	..	Case seen nine years after injury
Complete anæsthesia of soles; partial loss of sensation of rest of limbs, except front of thighs. Sensation better on inner than on outer side of thighs. Sometimes felt passage of catheter.	At first retention, later, dribbling of urine. Retention of fæces.	Bedsore.	Absent.	Fracture of body and dislocation forwards of 2nd lumbar vertebra, the cauda equina being "lifted on a bridge of displaced bone."	
Anæsthesia of genitals, but condition of limbs not at first noted. Felt pain when catheter entered bladder. On 8th day had perfect sensation in fronts of thighs. On 39th day had sensation in front of thighs and legs, in hypogastric region and scrotum; good sensation in 1st and second toes, partial in the others. Complete anæsthesia of back and inner sides of thighs.	Retention of urine. Involuntary defæcation. Urine ammoniacal for a time. On 39th day had desire, but no power, to pass water.	No priapism.	Prominence of the spine "in the lumbar region."	..	From the distribution of the anæsthesia, and from the position of the spinal prominence, the author thought it probable that the lesion affected the 4th or 5th lumbar vertebra.

No.	Reference.	Sex.	Age.	Result.	Probable Lesion.	Paralysis, &c.	Reflexes.
6.	Hutchinson, 'Lond. Hosp. Rep.' vol. iii. p. 332.	M.	33	Partial recovery in three months.	Dislocation of 2nd lumbar vertebra.	Paralysis of lower limbs, followed by some wasting, especially of glutei.	No note.
7.	M'Donnell, 'Dublin Quart. Jour. Med. Sci.' 1866, vol. xlii.	M.	31	Partial recovery in twelve months.	Upper lumbar.	At first, paralysis of lower limbs. At end of second month, complete paralysis below the knees, and very little power in the thigh muscles except the sartorius.	No reflexes below knees. Exaggerated in thighs.
8.	Leyden, 'Klinik der Rücken-marks-krankheiten,' vol. ii. p. 143.	M.	45	Death in seven weeks.	Fracture dislocation of 1st lumbar vertebra.	Lower limbs were paralysed, but some power remained in adductors and anterior muscles of left thigh. After three weeks there were cramps.	"Not increased."
9.	Leyden, 'Klinik der Rucken-marks-krankheiten,' vol. i. p. 340.	M.	32	Death in five months.	Fracture of 12th dorsal and 1st lumbar laminæ.	Lower limbs almost completely paralysed, but had some power of rotating and adducting thighs, and attempts at flexion of knees. Complete passive flexion of knees prevented by spasm of quadriceps. Adduction and inwards rotation of left thigh. Occasional cramps and tremors. Muscles of legs and back of thighs did not react to farad. current; but those of front of thighs did so readily.	No note.

Anæsthesia.	Bladder and rectum.	Vaso-motor and Trophic.	Local Symptoms.	Post-mortem.	Remarks.
Anæsthesia of scrotum, penis, and urethra; partially of thighs; completely of legs. At interval of three months, anæsthesia of feet, buttocks, and perineum; numbness of penis, scrotum, and urethra; fairly good sensation in thighs; better sensation on soles than on dorsum of feet, and on inner than on outer side of leg.	Retention of urine and fæces.	..	Projection of 3rd lumbar vertebra.		
At first anæsthesia of lower limbs. At end of two months had anæsthesia of feet, obscure sensation from ankle to knee, especially on left side; hyperæsthesia of thigh, especially on right side.	Retention of urine, followed in four or five days by incontinence. Ammoniacal urine.	Occasional priapism. Wasting of lower limbs. Perspiration of feet and ankles. Œdema of penis and scrotum. Urethritis, cystitis. Bedsore on back.	An immovable projection 4 inches above level of umbilicus.	..	The spine was trephined, which was followed by some improvement in the symptoms.
Anæsthesia of lower limbs, except from front of left thigh to dorsum of foot, the loss of sensation extending as high as the buttocks and sacrum, affecting also the penis and urethra; but less marked on the front than on the back of the thighs. Hyperæsthesia in inguinal region, shooting pains in limbs and pain in bladder.	Retention of urine and fæces.	No erections. Sweating of feet; œdema of lower limbs and scrotum. Bedsores of feet and sacrum.	No note.	Fracture of 1st lumbar vertebra, the cartilage immediately below which projected backwards some ¼ in. (7 mm.). Lumbar cord swollen, soft and pale. Other secondary lesions.	See text. This is given by the author as a typical case of traumatic myelitis.
Anæsthesia not complete, but sensation almost lost in the feet, very obscure in legs, back of thighs, and buttocks; better in front of thighs.	Retention of urine and fæces.	Slight œdema of legs. Bedsore on sacrum.	Spinous process of 1st lumbar vertebra projected backwards and to the right.	Fracture of 12th dorsal and 1st lumbar laminæ, and of body of 1st lumbar vertebra; tear of dura mater; compression of cord and cauda equina. Inflammation of pia mater and softening of cord extending to upper level of lumbar regions. Secondary lesions.	Death from uræmia.

No.	Reference.	Sex.	Age.	Result.	Probable Lesion.	Paralysis, &c.	Reflexes.
10.	Hamilton, 'Dublin Quart. Jour. Med. Sci.' vol. vi. 1848.	M.	25	Death in two months.	Fracture through body and laminæ of 2nd lumbar vertebra.	Paralysis of lower limbs.	No note.
11.	Hutton, 'Dublin Jour. Med. Sci.' vol. xxi. 1842.	M.	..	Death in six weeks.	Fracture of 1st lumbar vertebra.	Loss of power of the lower extremities.	No note.

urine, and involuntary defecation. In about six months the paralysis passed off, but the retention of urine was followed by incontinence, which, together with the rectal trouble, persisted. After death there was found some compression of the lower part of the spinal cord, and therefore necessarily of the surrounding nerve roots. The affected region of the cord showed degenerative changes. On these grounds the author concluded that this terminal portion of the cord contains the ano-vesical centre: a conclusion which appears not entirely warranted, in view of the fact that the cauda must have been somewhat compressed and that, as shown by our cases, the bladder troubles would then probably be the most persistent. We cannot, therefore, be certain that the case was purely one of cord lesion, uncomplicated by injury to the cauda.

In conclusion, I would draw attention to certain points in the diagnosis of those cases of pressure upon the cauda equina.

1. From locomotor ataxia. In a traumatic case there is

Anæsthesia.	Bladder and rectum.	Vaso-motor and Trophic.	Local Symptoms.	Post-mortem.	Remarks.
At first there was anæsthesia, except on the front of the thighs. In a few days this region was also affected, but again recovered.	Retention of urine, followed by symptoms of cystitis.	..	Projection of 2nd lumbar spine.	Fracture through upper part of body of 2nd lumbar vertebra and its laminæ, with compression of the cord "just above the cauda equina."	Death from erysipelas.
The "external parts of the lower limbs were quite insensible, the internal still retaining a considerable share of sensibility."	Retention of urine and fæces. Cystitis.	Bedsore on left buttock. The right tibia and fibula were also broken and united readily. Temp. of lower limbs was "at first" 62°, afterwards 95°.	Interval between last dorsal and 1st lumbar vertebræ.	An oblique fracture of the body of the 1st lumbar vertebra, with compression of the cord and cauda equina, which were bathed in pus.	

little likelihood of confusion, although even here it might possibly be regarded as ataxia consequent upon an injury. But in a case of tumour, or spina bifida, &c., the occurrence of a perforating ulcer, pains in the limbs, patches of anæsthesia, loss of knee-jerk, and some difficulty in walking, with or without bladder troubles, might well mislead the unwary. We must then note the absence of pupil symptoms, of girdle-pains, gastric crises, &c.; on the other hand, we shall find the peculiar distribution of the anæsthesia as above described, the affected muscles will be more wasted than in ataxia, and may present the reaction of degeneration, and there will be loss of power as well as inco-ordination. The gait differs from that of ataxia, and is characteristic, there being not only clumsiness and sprawling, but marked dropping of the toes.

2. From injury or disease of the lower part of the spinal cord. Here we must be guided by the exact site of the local symptoms, if any be present, remembering that the cord does not extend below the lower border of the first lumbar vertebra.

Also, if the cord be there injured, there will probably be some anæsthesia or hyperæsthesia of the last dorsal or first and second lumbar nerves; and again we may expect more rapid trophic lesions than in injuries of the cauda. If the symptoms be asymmetrical, we have probably to do with an affection of the cauda rather than of the cord. Whether in a partial crush above the level of the last dorsal vertebra, the damage done to the anterior crural nerves bears the same relation to that done to the sciatics as in a crush lower down, I have at present no evidence to determine.

3. From extra-spinal nerve diseases and injuries. We have here several difficulties to contend with, but in most instances a careful consideration of the case will lead to a correct conclusion. The only disease liable to be mistaken for an affection of the cauda equina is some variety of multiple peripheral neuritis, and here we may usually decide the point by finding some affection of the upper limbs, by the marked preference of that disease for the extensor surfaces and by the slighter sensory symptoms. In cases of injury the difficulty is greater. Even the limitation of symptoms to one side of the body is not an absolutely pathognomonic sign, as is indicated by Mr. Erichsen's case (No. 1, table). Nevertheless, complete unilateral distribution or perfect symmetry would be strong arguments for the lesion being respectively outside or inside the spinal canal. We are again aided by the seat of any local signs of injury; and finally we might with certainty pronounce the case to be an affection of the cauda, if we found the distribution of the sensory and motor symptoms to accord closely with the above-described types.

I have but one more remark to make here, and that is with regard to treatment. In cases of paralysis from pressure upon peripheral nerves, it is now an established rule of practice to free the nerve by a suitable operation, and in cases of division to place the separated ends in suitable position for re-union. Success is almost invariably the result of such action, and perfect recovery has followed after periods so prolonged as eighteen months.[1] Now the cauda equina is simply a bundle of peripheral nerves. Hence then, if it be compressed, or even

[1] Reg. Harrison, 'Brit. Med. Jour.,' 1886, vol. i. p. 443.

partially torn, by an injury to the bones of the vertebral column, we may expect to relieve or cure our patient if we can take off pressure and remove any cicatricial tissue in which the nerves may be involved. So much must, it appears to me, be granted by all, for whatever view may be taken of the power of regeneration of the spinal cord, there can be no two opinions of that of peripheral nerves. Again, we may probably assume that, where an injury to the cauda equina is followed by persistent symptoms, there is persistent pressure either by means of displaced bone or cicatricial tissue. Hence, in these cases there is a removable obstacle to recovery. If there were no such "gross" obstacle, recovery should occur, as in injuries of other peripheral nerves. It has been very properly urged that, in injuries of the spinal cord itself, we cannot distinguish the effects of a transient from those of a permanent crush, and that we should not "trephine," because we can rarely be sure of finding any bone pressing upon the cord. But this objection does not apply to injuries of the cauda equina, because the mere fact of recovery not ensuing in the course of some weeks is evidence of continued pressure.

Thus then we may say that, in injuries of the cauda equina which do not spontaneously recover, there is probably either a cicatrix or a piece of displaced bone which is keeping up the mischief, and that if we remove this noxious agent we may very confidently hope for a cure. Are we then justified in operating for its removal? I am strongly of opinion that we are. The operation itself is certainly not to be compared in difficulty with many which are daily undertaken without hesitation. The only special danger is that of meningitis, and with modern antiseptic precautions I see no reason to fear this, especially if we delay operating until there has been time for the formation of meningeal adhesions around the seat of injury. In most cases it will be found unnecessary to open the meningeal sheath at all, the source of trouble being merely a fragment of bone, or, as in Mr. Jones's case, an extra-dural cicatrix.

On such *à priori* grounds I would advocate trephining in cases of injury to the cauda equina, always bearing in mind the following conditions.

1. We must be sure of the localisation.

2. We should, if possible, wait for a reasonable period—say six weeks, before operating, and should then do so only if the patient shows no signs of spontaneous recovery.

3. Should the paralysis of the bladder be early followed by severe cystitis, and should we suspect secondary renal troubles, what are we to do? On the one hand, not to operate probably means death; and on the other, the risk of operation is enormously increased. Further experience may enable us to settle this problem, but at present the outlook appears to be sufficiently gloomy. A third course has suggested itself to me, but I have had no opportunity to test its results, viz. to drain the bladder by means of a supra-pubic cystotomy. The object of draining the bladder is obvious, and the supra-pubic appears preferable to the perineal route, because we are thereby enabled to construct our fistula through parts which are not anæsthetic and predisposed to slough.[1] If we could thus prolong life for a time, we might afterwards proceed to operate upon the spinal column, with fair hopes of a successful result, and encouraged by the great improvement effected in the case of R.M.C. and the cure obtained in a case recently published by Lauenstein,[2] in which there was complete paralysis of the lower limbs, etc., from a dislocation of the twelfth dorsal vertebra, and where a perfect cure resulted from the removal of the displaced arches. It would appear, therefore, that whether "trephining the spine" is or is not justifiable in cases of injury of the cord, it is certainly the proper treatment to pursue in those of the cauda equina, and hence the importance in such cases of making an exact diagnosis.

Postscript.

The above paper was written in June 1887, since which date there has appeared in the 'Lancet' for July 2nd, 1887, a most interesting article by Mr. Bland Sutton on "Spina

[1] It is hardly necessary to point out that, in an injury to the cauda equina when the symptoms of shock, &c., have passed off, the chief danger to life lies in the occurrence of bedsores or urinary troubles.

[2] 'Centralblatt f. Chirurgie,' 1886, No. 51.

Bifida Occulta." The author quotes one case of his own and others, previously published, illustrating the relationship of this affection with perforating ulcer and pes varus; but as there is no complete account of the nervous symptoms in these cases, I can only suggest, without being able to demonstrate, that they are probably similar to those above described.

FOLIE À DEUX.

BY D. HACK TUKE, M.D., F.R.C.P.

CASES of *Folie à deux*, for which we have not a good corresponding English term, are sufficiently uncommon to warrant my recording some interesting examples, and making them the occasion for a few observations. "Communicated Insanity" is the best term I know of, but it does not cover those cases in which the disorder is not strictly speaking communicated, and which the French include. "Double Insanity" is one synonym, but then it must be understood that I include under this head cases in which a greater number of persons become affected.

It is important to understand clearly what we mean by the phrase *Folie à deux*. Is the element of contagion, in the sense that fear is contagious, an essential part of the definition, or is it sufficient that two or more persons become mentally affected at the same time, and when together, by means of the same exciting cause? Or, again, may we stretch the definition to include the insanity of twins when they become insane contemporaneously and are not living together, on the supposition that there is a special sympathy between them?

Now while I should place in the first rank those cases in which there may be fairly said to have been contagion, in the sense that Fear is contagious, or that we speak of the contagion of a good or bad example, I include under the term Double Insanity, not only :—

(1.) Cases in which A. B., being insane, affects C. D. in consequence, and infects him with the same mental disorder ; but—

(2.) Cases in which C. D. becomes insane from companionship with A. B., not in consequence of the direct transference of morbid ideas, but in consequence of the shock or strain arising

out of the painful impressions produced by witnessing the attack, or by the strain of nursing a patient.

(3.) Cases in which two or more persons become insane simultaneously from the same cause.

(4.) Cases may even be included in which one lunatic infects another lunatic with his special delusions.

(5.) Twins.

The following are, briefly, the particulars of two cases I was recently requested to see, and are good illustrations of the *first* class.

The patients, neither of whom had an insane inheritance, and were not related, were a gentleman and his wife, the former being a partner in an extensive business in the City. The latter was an Irvingite, and the prominence given by the Church of which she was a member must not be altogether overlooked in determining the causation of the attack of mental disorder. In the next place, a gentleman, also an Irvingite, paid Mr. and Mrs. —— a visit, and induced them to try some experiments with the Planchette, which were regarded as very successful and remarkable. At the same period, Mr. ——, I may mention, had a difficulty in making his business accounts come right, and having discovered the error, or believed he had discovered it, he attributed his success to a spirit called "Minnie." Mrs. —— was, however, the first to be carried away by the notion of spirit influence, although the strongest-minded of the two, and, as far as I can discover, it was she who drew her husband in the same path, rather than the Planchette directly. Anyway, when I saw them, the lady was the active ruling influence, and the gentleman was the passive element, and the willing subject. I found them both in bed. The former was excited, and poured forth a stream of words. She said that spirits were constantly speaking to her, that part of their conversation referred to stabbing, and that they were cognizant of my presence in the room, and said many things to her about me, on the whole favourable, I am glad to say. She informed me she had been impelled to use bad words. She stated, in answer to my inquiry about the *sound* of the voices, that their voices were not so loud as mine, but that she had become possessed with a sort of instinctive

sense of hearing. She heard voices with *both* ears, and their loudness was not affected by closing them. When I assumed that she was a believer in modern spiritualism, she indignantly denied it, said she hated it, and wished to be rid of these voices. While we were talking, one of her children was crying downstairs, upon which she remarked that the spirits told her that some one was torturing the child. But the most interesting feature of Mrs. ——'s auditory hallucinations was this: that she heard her own words, as she spoke to me, *repeated* audibly and distinctly.

As regards the visual sense, she related to me having had a vision in the night when awake.

The olfactory sense was distinctly affected. She complained of a smell of death—"like vaults, you know."

I could not clearly make out any gustatory hallucination.

General sensation was normal. There was no anæsthesia whatever.

Physically, her countenance was flushed, her head hot; the skin was moist and warm. The temperature in the axilla was 100°, the pulse 90, and full. The tongue rather dry. The pupils were somewhat dilated, and re-acted well to light. Bowels confined three days.

I should mention that her left hand was bandaged, as she had cut it in thrusting her hand through the window in the night in order to keep the devil out. In fact her excitement was so great, that the governess barricaded her bedroom door, and a servant fled from the house in her night-dress, and took refuge in the nearest signal-box on the railway. Whether the signals were neglected that night I have not been able to ascertain.

Having examined this patient, whose form of mental disorder may well be called that of demonomania, I passed to the other side of the bed, and pursued my enquiries into the condition of the husband, who during this time had maintained silence, although he had been much excited in the morning, the wife (I should have mentioned) having been at the same time in a sort of stupor for many hours.

On conversing with Mr. —— I found him fairly calm, but possessed with the same delusions and hallucinations as his

wife. He told me he heard voices ordering him to do certain things. He said he believed that they had administered strychnia to him. He assured me that a clerk in his business was a medium, and exerted an influence upon him and his wife.

It was interesting to note that, while she complained of entire sentences being immediately repeated, he stated that the latter part only of each sentence was repeated.

With regard to the *visual* sense, he, like his wife, saw, as he said, the red sun in the room in the middle of the night. Since his recovery he has informed me that during this attack he could see with exquisite vividness the railway and the trains pass his house, at a distance which prevented him seeing them at all clearly when he was well.

Like her, also, he suffered from hallucinations of *smell*, and was free from those of *taste*.

His physical condition does not call for remark. His pulse was 80, his tongue moist, his temperature normal. His bowels were constipated. The pupils acted well to light.

On my advising him to go into another bedroom, he said he did not like to do so, as the gentleman who had been staying with them having occupied it, there were evil spirits remaining there.

These mental symptoms had manifested themselves for less than a week, and the malign influence of the visitor had been exerted for some days previously. The wife appears to have been the first to become affected, and, as I have already said, was the one to impress the husband with a belief in the reality of those hallucinations.

These patients remained together, notwithstanding my strong recommendation that they should be separated; yet they improved under treatment, and eventually recovered after a change to the seaside; but the husband at the present time, although attending to business, confides to me that whenever he is tired, the voices return, often addressing him in very unparliamentary language for not listening to them; but, he adds, "*there is no fear of my doing so, and I do not admit hearing them to any one.*"

It may be open to doubt whether the following is an

instance of Folie à deux, or whether it was a mere coincidence that the symptoms of one patient resembled those of the other, but the case is interesting.

A gentleman of large means became possessed of the idea that his food was tampered with. Labouring under this suspicion of poison being placed in his food, he would secrete a piece of bread during dinner, and when he retired to his study would carefully examine it. In this way his collection of morsels of bread was often very considerable, and on one occasion when his house was broken into at night, the burglars opened his escritoire—only to find a collection of stale bread. This gentleman moved in society, and frequently took the chair at public meetings, and very few were aware of his peculiar delusions. Among those who did know of them were the servants in the establishment, including the coachman. He was a highly-respectable and steady man, and loyal to the backbone to his master. His mind gave way without any shock or worry, and the form of mental disorder was that of suspicion of poison in his food. When called in to see him I could not fail to be struck with the resemblance between the mental symptoms of the coachman and his master, and I need hardly say that I should not have commented on it to any member of the family. But the gentleman's own daughter spontaneously made the remark: "Why he has got the same delusions as those which have affected papa." In some of the other symptoms she also detected a resemblance.

I repeat that this occurrence may have been only accidental, but at any rate I think it quite possible that, while the coachman's attack of insanity was quite independent of that of his master, he, when he broke down, dwelling moodily upon his condition, he bethought him of those delusions of poison by which his master had been possessed, and began to give credence to them, and then to suspect that he himself was the object of the same malign influence.

Having observed in the last 'Report of the Isle of Allan Asylum' the admission of a man, his wife, and their daughter on the same day, I wrote to the Superintendent, Dr. Richardson, for the particulars, and, thanks to his courtesy, I am able to give them here.

The father, William Cairn, admitted Feb. 26, 1886, was 70 years of age, a farmer, and believed himself to be pursued and persecuted by the whole House of Keys; that he was the owner of extensive property, out of which he had been kept by that House and the high bailiff. He asserted that mobs had been raised to destroy his houses and cut down his trees. He had, he said, been assaulted by the men who had robbed him, with crowbars and pickaxes; when he endeavoured to obtain redress of these grievances, he had been prevented by telegrams and ghosts.

His wife, ten years younger, asserted that her property had been sold against her will; that she had telegrams from invisible wires to say she must hang herself in consequence; and that her neighbours had put blood on the door and over the house.

The daughter of these people, admitted on the same day, was 26 years of age; was silent and morose, with the exception of saying "first-rate" to enquiries about her health. Her mind, in fact, was too demented to allow of her entertaining the delusions of her parents. How long she had been affected is not stated, but Dr. Richardson informs me that she had returned home from service some time previously, and he is of opinion that the insane ways of her parents had much to do with inducing her present condition of mind.

As to the man and his wife, the first symptoms arose about sixteen years ago after the loss of a little farm. They began to think they were entitled to property of great value, and eight years ago they went to London to Somerset House, to establish their claim, and have, their relatives say, spend "many a bright pound" in their search after the imaginary wealth.

Last year, in France, a whole family, consisting of six persons—the father, mother, two sons, and two daughters—were simultaneously attacked with demonomania. Dr. Lapointe, who reports the circumstance, states that they were orderly people, economical, temperate, and were generally esteemed. They were, however, hypochondriacal, and possessed of mystical ideas. They gradually came to believe that they were poisoned by sorcerers; the devil was in their clothes; they constantly saw him. They desired to be freed from his presence by exorcism; they regarded themselves as lost, and gave themselves up to many eccentric practices. At last they became dangerous, wandered about, and attacked the

peasants, so that it became necessary to place them in an asylum. At the end of a fortnight they were discharged from the asylum, apparently recovered. During two years they resided on the farm which they cultivated. Then, suddenly, the mother gave the signal of an attack, and the same ideas were reproduced in all the members of the family. It became necessary to place them once more in an asylum. It may be added that another member of the family, a son, being from home, was by this means saved from an attack of insanity.[1]

Here is an instance of *Folie à quatre*, reported by an Italian physician.

A woman, aged 47, was the daughter of a man who, having been a foundling, devoted part of his life to trying to discover who were his parents. In consequence of constantly hearing her father speak of his mysterious origin, she began to think he must be descended from some one of noble birth, whose fortune she would some day inherit. This idea, which became an insane delusion, she communicated to her mother, and also a lady in whose service she was as *femme de chambre*. This lady systematised the delusion still further, and believed that the rich ancestor of her protégée was a general in the First Empire, and that he had bequeathed a million francs to the hospital at Milan. Of course all the pains she was at, all her negotiations, ended in nothing, and all her statements were disputed.

Strange to say, the principal actor in this series not only married, but succeeded in conveying the delusion to her husband. Thus four persons were all inoculated with the same ideas, and, as might be expected, these were followed by hallucinations and suspicions of poison. In vain were they separated; they remain insane at the present time.[2]

When two lovers agree to commit suicide together, we may perhaps be in doubt whether they are, strictly speaking, insane. Still the fact serves clearly to illustrate the influence exerted by one mind over another in regard to a morbid train of ideas, and ideas of sufficient force and intensity to occasion desperate deeds. The following cases of simultaneous suicide, not of lovers, may be fairly regarded as illustrations of *Folie à deux:*

In May last, two young ladies of Munich, the Baronesses Anna and Louisa Guttenburg, aged respectively 26 and 23, committed

[1] 'Annales Médico-psychologiques,' November, 1886.

[2] "Archivio Italiano per le malatie nervose," &c., xxi. fasc. 5 and 6.

suicide by drowning themselves in the Starnberg Lake, on the identical spot where the King of Bavaria was found dead eleven months before. It is stated that they had held many melancholy conversations on the tragical fate of the King, and repeatedly went to the Lake in order to throw flowers into his watery grave. At last they gave way to the impulse of following the King's example, and taking a boat to the spot, which is close to the shore, they dropped silently into the water. Next morning, when the missing boat roused suspicions, a search was made, and they were found in the soft clay, firmly clasped in each other's arms. They were both pretty, highly cultivated, and rich.[3]

I think that a tragedy which occurred recently in Vienna may be properly placed under this division.

A lady lost her husband some years ago. She then begged an elder unmarried sister to live with her. In the course of time, the grief of the widow turned her head, and she became quite insane and was placed in an asylum. Up to this point of the history we have not an example of *Folie à deux.* It was the grief of the widow upset the sister's mind. Whether they belonged to an insane family is not stated. But now comes the portion of the story which bears on the subject of my paper. Last Christmas the insane sister was discharged from the asylum, recovered. The result was, that after her return to her widowed sister she not only relapsed, but infected her sister, and at length they resolved to commit suicide. They took a cab and drove to the foot of the Kœhlenberg, where it rises abruptly from the Danube. Having dismissed the cab, they were seen sauntering along the bank of the river. Shortly after, a railway guard saw a female figure floating on the water. Having taken a boat, he succeeded in getting the body out. He then discovered that a second woman was fastened with cords to the back of the first. The woman at the top was the insane sister, and was restored to consciousness after a short time, and recovered. The other sister—the widow—was dead. While this was being ascertained, the sister who had been saved ran to the river and threw herself in again, but was again rescued. She was sent back to the asylum, from which she had probably been too soon discharged.

Having given striking examples of the first class of cases in which contagious sympathy exerts a great influence, I proceed to give an illustration of the *second* class of cases, those,

[3] 'Daily News,' May 15, 1887.

namely, in which an insane patient caused the attack of insanity in another from shock, or strain.

I was consulted about a married lady in consequence of her having become mentally deranged, the form of the disorder being melancholia with suicidal tendencies. The birth of a child—the first—and a good deal of domestic anxiety appeared to be the causes of the attack.

A sister of this lady was summoned by a telegraph to join her. She had attended her in her confinement, was not in strong health, and was somewhat unnerved, as was but natural, by being sent for to her sister on account of illness. She was by nature quiet and undemonstrative, and did not betray any undue emotion; so that the patient's husband had certainly no idea of there being anything the matter with her when she came to see her sister. She arrived on a Tuesday; she was alone with her till the Friday. Then a mutual friend went over by accident to see them, and Miss ——, the visitor, shortly said, "I must go home at once, or I shall go mad." A medical friend attributes this to the shock of finding her sister insane.

She left the same day, travelling home alone. The mutual friend, finding her quite as insane as the patient, was very uneasy at her making the journey by herself. From the time she reached home she was in a state of extreme mental depression, becoming at last much worse than her sister. About two months later she made a serious attempt on her life, by cutting her throat with a razor. She was then removed to an asylum, and for several months was not expected to live. She was ill for at least a year, rarely uttering a word and profoundly depressed. Recovery followed, and she has remained well.

The sister who was first attacked, and was my patient, never passed into so bad a state, and it was not necessary to place her in an asylum. She recovered perfectly.

In regard to these cases (those in which visiting, nursing, or taking charge of an insane patient induces a prejudicial mental effect) it is of course difficult, if not impossible, to decide how much is due to imitation and sympathy, and how much to the mere strain from constant attention to the patient. It is remarkable that attendants in asylums do not

break down mentally oftener than they do. Indeed, seeing that some attendants are bound to become insane whether attendants or not, I think the fact that attacks of insanity in this service do not attract special notice is a proof that the contagion of insanity is somewhat rare.

Without pretending to accurately determine, in the following case, how much was due to mere companionship and how much to the responsibility of having charge of the patient, I think it is worth mentioning in this connection.

Miss ——, a young lady acting as constant companion to a patient of mine in lodgings for a period of some months, became the subject of mental symptoms herself. The patient had delusions, but her companion became simply depressed and hypochondriacal, without any delusions. She went into a medical man's house, and becoming worse, desired to be admitted into an asylum. I do not think there was any transference, in this case, of insane ideas from the patient to her companion, but that the care of the case was the real cause in a lady who was predisposed.

In a very sad case about which I have been recently consulted, I have no doubt of the influence exerted by an attack of insanity in a lady upon her sister who was with her at the time. The latter felt the shock of the event deeply, called in question the necessity of placing her sister in an asylum, and became alienated from other members of the family. She could not sleep, and was unable to pursue her avocations. A near relative had previously been insane, but there was no evidence of heredity.

My *third* class comprises cases in which two or more persons become insane simultaneously from the same cause.

The most familiar illustration of this class is the effect produced by the Revivalist. The immediate excitement, and even the hysterical convulsions induced, are conditions which cannot be ignored in their bearing upon the influence which may be exerted by that rapid spread of sympathetic feeling which we recognise as epidemic in our own days, as well as in the Middle Ages, although they may pass off without constituting actual insanity. But the effects are sometimes, unfortunately, permanent, and constitute then striking examples

of *Folie à deux* or a larger number. I shall not occupy space in bringing forward cases falling under this category, but proceed to the

Fourth class of cases, those namely, in which one insane patient infects another lunatic with his or her delusions. That this is a much rarer event than one might have expected will be admitted by any one who has endeavoured to collect examples. Laségue regarded this occurrence as very rare. However, such cases do from time to time occur, and I will briefly cite one or rather two I have seen in Bethlem Hospital, which were under Dr. Savage's care.

Mrs. S. was admitted into the hospital on March last, labouring under delusions, the most prominent being that she was infected with syphilis, which, by her touch, had infected all others, far and near, even her most gracious Majesty. No evidence of syphilis could be obtained.

When in the hospital she was depressed, and talked freely about her delusion. She was frequently with Mrs. A., a patient in the same ward, and after awhile it was found that the latter was possessed of the same delusion in regard to herself. On the 18th of June last I specially questioned her on this point, and found that she fully believed herself to be labouring under syphilis. She interpreted some tonsillitis, which she had, to be a syphilitic sore throat, and referred to other symptoms also as being due to the same cause. I could not make out that she thought the other patient had infected her. It appeared that she had simply adopted her delusion. I may add that, on her admission, she was labouring under great mental depression and intense suicidal tendency, and had auditory hallucinations.

Although cases of this kind undoubtedly occur occasionally, the general disposition of the insane is to hold tenaciously to their own delusions, and to smile at the delusions of their comrades in disease.

The *fifth* and last class has reference to the insanity of twins.

I saw an example in the York Asylum, some years ago, of twins in a state of acute mania at the same time, but I cannot recall the particulars. There have been some marked cases at Bethlem Hospital in which a very similar course has been run

by twins. It really seemed as if there was a sort of sympathy between them, or perhaps it would be more correct to say, that two constitutions having originated and been built up at the same time, and under precisely the same circumstances, they were so nearly identical that the exciting causes of insanity produced, when operating upon them, the same result. The coincidence was curious, that such causes should happen to come into operation at the same time.

There is now a man in Bethlem in an advanced condition of general paralysis, whose twin brother has been under Dr. Clouston, at the Moningside Asylum, as a general paralytic. There was not a long period between the invasion of the attacks in each. Dr. Clouston's patient is dead, Dr. Savage's is in the third stage of the disease.[1]

Some time ago there were twin sisters in Bethlem, whose histories ran a singularly parallel course, although not together.

There were admitted in March, 1880, into the Southern Counties Asylum, Dumfries, twin sisters, Eliza and Isabella Scott. I have obtained from Dr. Rutherford copies of the certificates. Unfortunately the other particulars are extremely scant. They closely resembled one another; were 23 years of age, single, and domestic servants. No other members of the family had been insane. The attack dated in both from the same day—ten weeks before admission. Both are stated to have had a previous attack of insanity, and also at the same period, viz. when they were 19.

The cause was not known.

They were suicidal, and dangerous to others. Eliza was suspicious of being poisoned, and had delusions about her friends. Isabella refused her food. Both were in a state of great excitement and incoherence. Isabella alternated between frenzy and depression.

Eliza's excitement passed away in about a week, and she was discharged, recovered, in rather less than two months.

Isabella did not recover so quickly, but she was discharged, recovered, in about nine months. She subsequently married; her recovery was apparently complete.

[1] The patient has died since this was written.

To these cases I would add a few observations. Under whatever head the cases may fall, it is important in studying them to have in mind these among other questions:—

(*a*) Are the patients members of the same family?

(*b*) Is there an insane inheritance, or, if not, is the patient, who appears to be the passive subject or parasite, distinctly neurotic? The question of predisposition is, for obvious reasons, highly important.

(*c*) How soon after A. and B. met did B. manifest mental disorder?

(*d*) How long did they continue to associate?

(*e*) To what extent did the passive subject develop symptoms identical with those of the active agent?

(*f*) Were they separated, and if so, what was the after-history of the patients, more especially that of the passive subject?

Conclusions:—

(1.) I think that experience shows, that the influence of the insane upon the sane is very exceptional, except under certain conditions, which can be laid down with tolerable accuracy.

(2.) As an almost universal rule, those who become insane in consequence of association with the insane, are neurotic or somewhat feeble-minded.

(3.) More women become affected than men.

(4.) It is more likely that an insane person able to pass muster, as being in the possession of his intellect, should influence another in the direction of his delusions than if he is outrageously insane. There must be some method in his madness.

(5.) The most common form which cases of communicated mental disorder assume is that of delusion, and especially delusion of persecution, or of being entitled to property of which they are defrauded by their enemies. Acute mania, profound melancholia and dementia are not likely to communicate themselves. If they exert a prejudicial effect, it is by the distress these conditions cause in the minds of near relatives.

(6.) A young person is more likely to adopt the delusions of an older person than *vice versâ*, especially if the latter be a relative with whom they have grown up from infancy.

(7.) It simplifies the comprehension of this affection, to start from the acknowledged influence which a sane person may exert upon another sane person. It is not a long road from this to the acceptance of a plausible delusion, impressed upon the hearer with all the force of conviction and the vividness of a vital truth.

(8.) It is not easy to determine to what extent the person who is the second to become insane, affects in his turn the mental condition of the primary agent. My own cases do not clearly point to this action, but there have been instances in which this has occurred; the result being that the first lunatic has modified his delusions in some measure, and the copartnership, so to speak, in mental disorder, presents a more plausible aspect of the original delusion.

(9.) Is it possible that those who are frequently with the insane become more irritable than they otherwise would have been, without being actually insane? This has been asserted by a French physician, but I am not prepared to support it.

I would say, in conclusion, that the subject I have discussed, is one of practical importance as well as curious interest.

We ought to be alive to the considerable danger of one member of a family who becomes insane endangering the mental health of another member of the family. I should discourage a sister nursing for long an insane sister, though I have a patient at present who is to a large extent nursed by her three sisters, and this in a family where a brother has been under my care, and a sister and the mother have been insane. Their devotion is, however, so great, that they do not like to leave their sister in the hands of strangers.

Again, I think that we should discourage susceptible young women, and especially hysterical ones, from associating with persons having delusions, or even entertaining wild eccentric notions short of insane delusions.

Lastly, when such cases fall under our treatment, there can be no difference of opinion as to the importance of separating promptly the patients who are affected, and of changing the scene completely. The passive agent—the parasitic growth—will probably gradually recover in a fresh and more wholesome environment.

THE GROUPING OF THE CRANIAL NERVES.

BY ALEX. HILL, M.A., M.D.

Fellow of Downing College, Cambridge.

THE key to the arrangement of the nuclei of the nerves of the spinal cord is to be found in their segmental succession. I believe that the recognition of this anatomical fact is due to the late Professor Aeby. It has long been a favourite physiological axiom of Ludwig's, under whose direction various researches into the segmentation of the spinal cord have been carried out.

In my lectures as Hunterian Professor in 1885, I attempted to apply this principle to the brain, and to check the observations hitherto made as to the position in the cerebral axis of the nuclei of the several cranial nerves by a consideration of the distribution of these nerves in the segments of which the head is composed. It appears to me that this matter is of the greatest importance to neurologists, for a proper understanding of the constitution of the lower parts of the brain is impossible without a knowledge of the plan of segmentation of the head. On the other hand, the determination, by those who make a special study of the brain, of the position in it of the nerve nuclei, ought to throw light upon the difficult morphological problem of the segmentation of the head.

In all attempts at determining the lines of division between the segments of the head the cranial nerves have played a prominent part, and rightly, for no other constituents of the body so long retain traces of their origin. Nerves are the aristocrats among the organs, adhering with greater pertinacity than the rest to their traditional course and constitution. To a certain extent, no doubt, this is due to the fact that the nervous system is the first part of the body to be

developed, and obtains ontogenetically as phylogenetically to a certain degree of structural differentiation, before the modified organs for the control of which it exists, put in an appearance. A muscle is altered in form to meet the requirements of a change in occupation. The blood-vessels which supply the muscles adopt the course most suitable for providing for the nutrition of the muscle in the altered position in which they find it at the time of their development. The nerve, however, is formed before the muscles, and strikes out consequently for the original situation of its muscles for generations after this has been changed. Hence it comes about, that while muscles and the bones which support them lose their metameric significance, the nerves preserve the records of the animal's ancestry.

It occurred to me some ten years ago that, since the nerves are outgrowths of a central system, their nuclei of origin within this system ought to exhibit a metamerism more marked than even that of the nerve trunks. I failed to classify them, however, because, following in the footsteps of all the morphologists who had up to that time dealt with this question, I expected to find a division of nerve roots into two series (sensory and motor, or dorsal and ventral) only, of which of course in the case of the cranial nerves, one series might be incomplete. Trying, however, to account for the apparently eccentric origin and distribution of the *spinal accessory* nerve, I realised that all the way down the cord, the columns of cells which belong to the anterior and lateral horns are so distinct as to belong evidently to nerve fibres supplying different groups of muscles.

The distinction between anterior and lateral horns is more obvious in the fœtus than it is in the adult. What may be the morphological division of the muscles into two groups with which this is correlated it is at present difficult to say. In the course of their growth the muscles exhibit an arrangement into somatic and splanchnic, as well as into dorso-lateral and ventro-lateral groups. It is better perhaps, in the present paper, not to attempt to decide to which of these systematic arrangements the division of the motor cells in the cord into anterior and lateral horns belongs. It suffices that the dis-

tinction is all the way down the cord (except perhaps in the lumbar and cervical enlargements, where the number of cells in each horn being great, the two regions become confluent), so clear that it is impossible to doubt, that the anterior nerve roots as they leave the cord contain two sets of fibres of different origin and destination.

In the cervical region the fibres from the lateral horn constitute a separate nerve, the spinal accessory. In other words, the cervical cord gives exit to anterior and lateral root-fibres, as well as entrance to the fibres of posterior roots.

This greatly simplifies the problem of metamerism. Up to this time morphologists looked upon all motor nerve-roots as of equal importance. Each cranial motor nerve was regarded as the homologue of a spinal ventral or motor root. A consideration of the arrangement initiated by the spinal accessory nerve teaches us, that it takes two motor cranial roots to equal one spinal ventral root, for in their exit from the spinal cord, except its cervical region, the fibres derived from the anterior and lateral horns run together. Not so the nerves which issue from the axis of the brain. In this part the separation between the anterior horn and its nerves, viz. the hypoglossal, abducens and motor-oculi, and the lateral horns and its nerves, the spinal accessory, facial and motor division of the fifth is distinct and easy to recognise.

At the time when I came to this conclusion, I was not acquainted with Van Wijhe's paper,[1] in which he puts the matter upon an infinitely better footing than any observations in adult anatomy could do, by tracing the origin of the muscles of the head to two distinct mesodermic sources, the "somites" and the "lateral plates," for which different groups of nerves are destined. My argument amounted to nothing more than bringing the known facts of adult anatomy to bear upon the question of cephalic segmentation. Van Wijhe's researches are referred to in the paper of Gegenbaur, which forms the excuse for this article. It will be noticed that Gegenbaur finds difficulties in the way of accepting the

[1] "Ueber die Mesodermsegmente und die Entwicklung der Nerven des Selachierkopfes" (*Natuurk. Verhandelingen Koninkl. Academie, Amsterdam*, Deel XXII., 1882, Sep.)

complete homology of the third, fourth, and sixth nerves, which according to Van Wijhe belong to the first three somites respectively.

Secondly.—Gaskell's researches have shown that throughout the cerebro-spinal system we have also to reckon with a visceral root, which, as a rule, accompanies the anterior motor root, but in the case of the vagus and pars intermedia constitutes an almost pure nerve, leaving the system in company with the lateral motor and sensory roots.

Adding to the sensory root the three motor ones thus determined, we find that when we attempt to homologise cranial and spinal nerves, we must take four of the former to equal

FIG. 1.—GROUPING OF NERVE ROOTS IN THE CORD

1. anterior; 2. lateral; 3. visceral; 4. dorsal; *a.* anterior horny; *b.* lateral horny; *c.* Clarke's column; *d.* subs. gelatinosa Rolandi.

FIG. 2.—GROUPING OF NERVE ROOTS IN THE MEDULLA.

References as in Fig. 1 (*c.* Clarke's column is now swollen out into vagus nucleus).

one of the latter, since in the head the union of sensory and motor fibres does not take place just outside the central nervous system as it does in the spinal cord.

Charles Bell classified nerve roots in two divisions—dorsal or sensory, and ventral or motor.[1] I might speak of my

[1] Bell also observed that, while certain cranial nerves, namely the hypoglossal, abducens and motor oculi, follow the same line of origin as the motor roots of the spinal nerves, the *regular nerves*, as he termed them, the rest of the cranial nerves

classification as based upon the FOUR-ROOT THEORY, for I believe that each spinal nerve contains four sets of fibres, which in the case of the cranial nerves are more or less discrete.

Thirdly.—It is curious to notice the way in which the roots of the spinal accessory nerve, which has already divorced itself from the anterior motor nerve, the usual companion of a lateral motor root, gradually incline across the lateral column to join at the upper part of the medulla with the vagus and the glossopharyngeal. The nucleus of the vagus was recognised by Ross as the upper end of Clarke's column. The nerve arising from it no longer takes its exit in company with the anterior motor root, but with the posterior and the lateral motor. The change in grouping is shown at a glance in the accom-

---

are *irregular* in their situation on the brain. The fifth nerve he rightly regarded as the sensory nerve of the head, but the remaining nerves, fourth, seventh glossopharyngeal, par vagum and spinal accessory, he considered as forming, with the "external respiratory" and phrenic, a group superadded to the regular nerves on account of their having a common function—namely, respiration. Dr. Gaskell, in a paper published some nine months after mine ('Journal of Physiology,' 1886, vol. vii. p. 1), gives to Bell the credit of not only discovering the division of nerve roots into sensory and motor, but also of recognizing the third great division of cranial nerves. This is very patriotic of Dr. Gaskell, and at the same time to a certain extent, from his point of view, justifiable; for Gaskell not only recognises the division of the cranial nerves (as given in my paper) into somatic motor (going to the muscles of the somites) and sensory; lateral motor (going to the muscles derived from the lateral plates) and *visceral*; but further subdivides the latter into efferent nerves going to visceral muscles, &c., and afferent nerves from hypoblastic surfaces (the glossopharyngeal and part of vagus). If the fourth and phrenic are taken out of Bell's list and the motor part of the fifth added to it, his respiratory group of nerves agrees with Gaskell's "splanchnic" group. In other words, the nerves which Bell observed to arise regularly from the base of the brain belong to the dorsal sections of the cephalic segments, the nerves which he classed as irregular to the ventral sections. Instead, however, of nerves of respiration, Gaskell would call them the nerves supplying motion and sensation to the viscera and to the arches by which the viscera are supported.

No one can read Charles Bell's writings on the subject ('Anatomy of the Human Body,' vol. ii. p. 373) without seeing that, far from having any such morphological classification in his mind, he was attempting to obtain anatomical data for a most untenable theory as to the functions of the body. Gaskell's scheme includes all that is contained in mine, and at the same time goes beyond it in what has a most plausible appearance of being the right direction. Until further morphological data are obtained, however, it seems hardly desirable to discuss its probability.

panying woodcuts. In Fig. 1 these nerves are seen to leave the spinal cord between its anterior and lateral column, although the dotted line indicates that in the cervical region the spinal accessory runs out alone through the lateral column. Fig. 2 shows one nerve only, the hypoglossal, leaving the medulla between its anterior pyramids and lateral column (olive), while three nerves come out between the olive and restiform body.

In reading the accompanying abstract of Gegenbaur's paper, it will be observed that the nerves of the organs of special sense receive a very varied treatment at the hands of anatomists. The olfactory optic and auditory nerves are regarded as either segmental nerves, or branches of segmental nerves, or nerves *sui generis*, and therefore out of the discussion.

The organs of special sense are looked upon as either independent gill clefts, or formed in connection with gill clefts, or even formed out of gill clefts. Or again, of the three organs one is regarded as having a different origin, and its nerve therefore a different homological value from the others. To the writer of this paper it has always seemed necessary that the nose, the eye, and the ear, should be treated alike. It seems highly probable that these organs are the only three left out of a complete series of segmental sense-organs which at one time existed, and that the relation of each of them to the central nervous system depends upon the period at which it specialised and became consolidated into its permanent form.

The uncertainty of morphologists as to the light in which the organs of special sense are to be viewed is due, as it seems to me, to a complete misapprehension as to their relation to the central nervous system.

It can be most easily proved, although the quoting of examples and references would make this paper too long, that the central nervous system was in the first instance deposited in the neighbourhood of the sense organs, and subsequently withdrawn to a common central ground for safety and convenience of exchange of impulses. In the first instance, the central nervous system consisted of detached clumps of ganglionic matter at the bases of the sense organs. Then the

clumps were connected together by commissures. Finally, clumps and commissures were withdrawn to a central situation. In the case of the first two sense organs, specialisation had advanced so far at the time when centralisation of the nervous system set in, that elements which in all the posterior segments are withdrawn into the central nervous system, retain their original position at the base of the nose and eye.

It is very likely, however, that the spinal ganglia mark the position of vanished sense organs, and that the fundamentally bipolar cells of which they are composed are portions of the ganglionic matter which at one time lay beneath the sense organs.

In the retina we find three kinds of nervous element, minute bipolar cells (nuclear layer), plexus (inner molecular layer), and multipolar cells. The olfactory bulb consists, as I have elsewhere shown,[1] of the same three sets of elements. I am of opinion that the cells of the spinal ganglia are the direct homologues of the bipolar cells of the retina and olfactory bulb, while the other layers of these two organs are in the case of posterior segments withdrawn into the cerebro-spinal axis.

Undoubtedly the nerves of special sense are of segmental value inasmuch as it is unlikely that any single metamer contained more than one sense organ, although, as Gegenbaur most forcibly shows, this had no necessary connection with a visceral cleft; but whether it was a branch of the general cutaneous sensory nerve, or, as is more likely, the sensory nerve, a branch of it, we have no evidence to show.

It is possible that the part of the head in which the nose and eye are situated obtained its form at a period prior to the existence of any such metamerism as the visceral clefts and arches indicate.

[1] 'Plan of Central Nervous System.' Cambridge: Deighton & Bell, 1885.

ON THE RELATION OF THE CENTRAL NERVOUS SYSTEM TO THE ALIMENTARY CANAL.—A STUDY IN EVOLUTION.

BY J. BLAND SUTTON, F.R.C.S.

Hunterian Professor, Royal College of Surgeons, Assistant-Surgeon to the Middlesex Hospital.

FOR many months I have been closely engaged in studying the pathological anatomy of spina bifida. During the time my attention was directed to this matter, Prof. von Recklinghausen published an interesting and elaborate memoir on Spina Bifida in Virchow's 'Archiv,' Bd. 106, 1886. Not the least important part in that painstaking piece of work, is the section which deals with specimens illustrating the frequent association of certain forms of spina bifida (especially the form known as syringo-myelocele) with defects in the alimentary canal, such as umbilical hernia, atresia ani, and so forth.

Still more recently I have been engaged in studying the curious condition of the spine known as *spina bifida occulta*, and whilst still fresh from that study, a very remarkable fœtus came under my notice, which suggested still further inquiry. The fœtus in question will be fully described in the 'Transactions of the Pathological Society,' 1888; it may be briefly stated here that it was the subject of spina bifida occulta in the lower lumbar region. Associated with this defect was, among other things, an imperforated pharynx, a communication between the œsophagus and trachea, atrophy of the vermiform appendix and imperforate rectum.

The conclusions to which I have arrived from an investigation of this material, as well as a careful consideration of the embryological history of the cord and alimentary canal, are, I venture to think, of some degree of novelty and interest, and will be embodied in this paper.

Invertebrates are distinguished from vertebrates, not only in the absence of anything resembling a vertebrate column, but in the non-possession of any structure resembling a spinal cord. To my mind it is clear that the cord must be regarded as primary to the column, that is to say, the cord was first evolved, but the vertebræ are of secondary and much later development.

Of recent years embryologists have brought to light some very remarkable facts relating to the connection of the alimentary canal and the spinal cord, and these facts have become so well established that at the outset of my argument I shall be able to put before you a diagram illustrative of the fundamental arrangement of the alimentary tract, from which the vertebrates of the present day inherit theirs.

The spinal cord, as is well known, is formed by the coalescence of the medullary folds on the *dorsal* aspect of the notochord. The body walls are formed by the coalescence of the somatopleure of each side on the *ventral* aspect of the notochord. Early in embryonic life a layer, the splanchnopleure, is detached from the inner aspect of the somatopleure to form the walls of the gut, by a median ventral coalescence. The outer layers of the ventral laminæ form the body wall and are continuous with the outer layers of the medullary folds, which in like manner give rise to the cutaneous investment on the dorsal aspect of the embryo.

Each lamina which helps to form the cord and gut respectively is lined by columnar epithelium. Although in higher types the cells lining the canal of the cord are ciliated, whereas those in the gut are simple, nevertheless it is not difficult to find instances among vertebrates of ciliated cells in the digestive tract. It is a fact of some significance that, so far as embryological knowledge extends, no organ save the gut and the cord is formed by the union of lateral plates.

The valuable researches of Kowalevsky on Ascidians, demonstrating the connection between the central canal of the cord and the gut, around the caudal end of the notochord by means of the neurenteric canal, has since been extended to vertebrata in general, including man, by such competent observers as Kolliker, His, Balfour, Goette, Heape, and others.

A survey of the evidence enables us to represent the primitive alimentary canal as a U-shaped tube lined with a continuous columnar epithelium. Each limb ends at its cephalic extremity in a cul-de-sac. The ventral segment of the tube is widely open to the yolk sac. Occupying the flexure of the tube is the notochord and subnotochordal rod. Fig. 1.

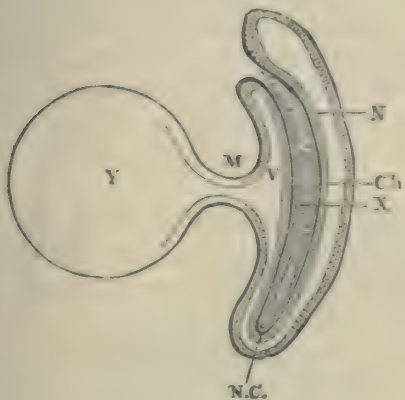


FIG. 1.—THE EMBRYONIC CONDITION OF THE ALIMENTARY CANAL AND CENTRAL NERVOUS SYSTEM OF A MAMMAL.

N. Spinal cord. N.C. Neurenteric canal. V. Intestine. M. Vitelline duct. Y. The yolk sac. CH. Notochord. X. Subnotochordal rod.

FIG. 2.—A DIAGRAM TO REPRESENT THE SUBSEQUENT MODIFICATION OF THE GUT.

S. Stomodæum. X. The pouch of Rathké. L. The diverticulum for the lungs. M. The vitelline duct (Meckel's diverticulum). G. Cloaca. A. Allantois. P. Proctodæum. * Luschka's gland, the persistent remnant of the post-anal gut.

The subsequent events in the ontogeny of these structures is interesting. The limb connected with the yolk sac may, for convenience, be divided into two sections—the fore and hind gut. The anterior end of the fore gut becomes connected with the exterior by an epiblastic involution—the stomodæum, which not only opens up the cephalic end of the gut, but has

close relationship with the cephalic end of the dorsal or neural tube by a diverticulum, the pouch of Rathké, which comes into union with the infundibular diverticulum from the primary encephalic vesicle. The coalescence of the stomodæum and fore gut results in the establishment of a communication between the pharynx and œsophagus.

An involution next appears in the caudal region—the proctodæum—which coalesces with the tube and establishes a nether opening—the anus. The remaining section of the primary tube on the ventral aspect of the notochord is known as the post-anal gut, represented in the adult by Luschka's gland, whilst the segment joining the ventral and dorsal portions of this primary tube is the neurenteric passage. All that portion of the tube on the dorsal aspect of the notochord anterior to the neurenteric passage becomes converted into spinal cord and brain.

Not only are the dorsal and ventral segments of the tube in connection by the neurenteric passage, but they are intimately associated in a way that is of some importance in support of my contention.

Occupying the flexure of the tubes is a chain of sympathetic ganglia which distribute branches to the ventral and dorsal sections of the tube. Those on the ventral portion of the tube ramify in the walls of the bowel, and are provided with intrinsic ganglia. These nerve plexuses are later recognised by the names of Auerbach and Meissner. Those distributed to the dorsal tube, ramify in its substance, and join with groups of nerve cells contained in the thickness of its walls, which constitute the essential elements of the grey matter of the cord.

A full and careful consideration of the facts briefly narrated in the preceding pages, has served to convince me that *the spinal cord and brain of vertebrata have been evolved from what was originally a section of the alimentary canal.* In other words, *the central nervous system is a modified piece of bowel.*

For convenience, the evidence in support of the view may be arranged in a series of paragraphs :—

1. The original continuity of the lumen of the gut and spinal cord.

2. The similarity in their mode of development and correspondence in point of time.

3. The relation of the lateral ganglia (sympathetic) to the walls of the gut and its intrinsic ganglia and to the ganglia (grey matter) of the cord respectively.

4. Each tube, the nervous and digestive, is protected by a serous membrane—in the one case the arachnoid, in the other, the pleuroperitoneal membrane.

5. In lower vertebrate forms the spinal cord is relatively larger than in man: in batrachians it much exceeds the brain in weight.

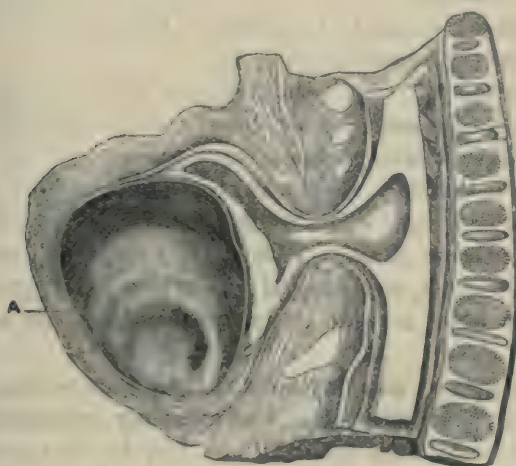


FIG. 3.—A COMBINATION OF SYRINGO-MYELOCELE AND MENINGOCELE—SYRINGO-MYELO-MENINGOCELE. (After Clutton, 'Trans. Clin. Soc.,' vol. xix.)

6. The association of certain malformations of the central canal of the spinal cord, especially *syringo-myelocele* and *syringo-meningomyelocele*, with malformations of the alimentary canal.

The last statement is of exceptional interest. Not long since, in dealing with certain forms of cysts, I ventured to state that, although we were absolutely in the dark concerning

the significance of the central canal of the cord, nevertheless, it was justifiable to regard it as an obsolete canal, that is to say, one that was functional in the ancestors of the present vertebrata, because, like such passages in general, it was extremely prone to take on cystic dilatation.

The general statement of the argument has not been over-burdened with facts, my object being not to obscure the conception. Strange as it may seem to look upon the central nervous system as a piece of modified bowel, the metamorphosis is not more remarkable than many others that could be mentioned.

Many who suggest modes of evolutions are always most anxious to indicate particular roads along which the modification may be supposed to have travelled. If it were necessary, suggestions could easily be made on this head, but it would not advance my argument one jot.

Speculations of this character are rare in pathological science; nevertheless, they are as fascinating, and perhaps may be as useful, in this as in any other department of biology. If employed judiciously, they may afford guidance of no mean value, provided those who advance such views do not attach to them a concrete value. Pathology, I am sure, can be of service to biology, and general views may be suggested by a study of morbid, as well as by the consideration of normal anatomy.

Finally, it is by such methods as these that we may hope to found a satisfactory classification of many pathological conditions which, at the present time, are, for the most part, arranged in a vague, arbitrary, and meaningless fashion.

ON A READY METHOD OF PREPARING LARGE SECTIONS OF THE BRAIN.

BY BYROM BRAMWELL, M.D., F.R.C.P. (EDINBURGH).

THE position and extent of tumours and other lesions situated in the interior of the brain are perhaps most accurately determined by cutting the organ into a series of transverse-vertical or longitudinal-vertical sections, after the parts have been fixed in position by some hardening agency.

The preliminary hardening is best effected by injecting the brain, as a whole, with Müller's fluid—a method which was, I believe, first recommended by Professor D. J. Hamilton, of Aberdeen.

With the object of facilitating the process of injection and allowing the fluid to distribute itself equally and evenly throughout the whole brain tissue, I am in the habit of using an ordinary stomach-pump syringe, and a "distributer" (see Figs. 1 & 2, pp. 436, 437), from which tubes pass to the four great nutrient arteries (vertebrals and common carotids).

Each of these tubes is attached to a canula (see Fig. 2, A), which is firmly tied into the artery. In order that this attachment may be easily made, it is necessary, in removing the brain, to see that the vessels are cut as long as possible. The canula should be attached to its tube before being tied into the vessel; and all four tubes should be tied into the arteries before they are attached to the distributer.

Before proceeding with the injection, I find it advisable to place the brain in a large wide-mouthed jar filled with Müller's fluid. The fluid in which the brain is immersed should be changed (by means of the syringe-syphon arrangement described below) and the brain reinjected on the following day. After an interval of two days the process should be repeated; and a

further injection may be made later, if it is thought desirable. The fluid in which the brain is immersed should be changed at least four times.

At the end of a month or six weeks, the organ is sufficiently hard to be cut into sections.

In those cases in which it is necessary or desirable to cut into and examine the brain in the fresh condition, the organ should be cut into a series of thick transverse-vertical sections,

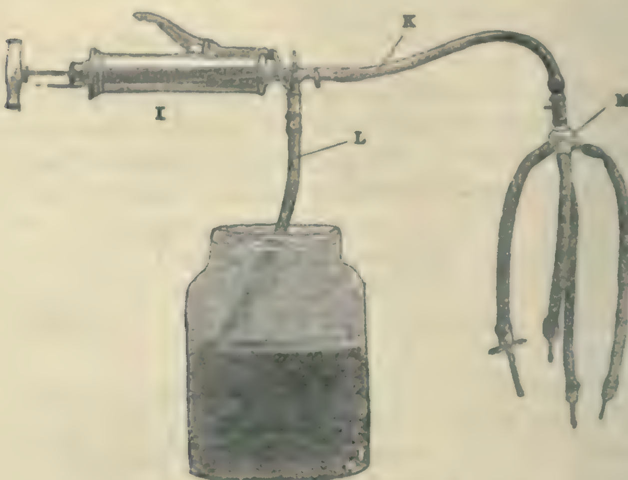


FIG. 1.—INJECTION APPARATUS DESCRIBED IN THE TEXT.

The letter I points to the syringe: L, to the supply tube of the syringe: K to the discharge tube, by which the fluid is conveyed to the distributer M, and thence to the four great vessels (vertebrals and common carotid arteries).

The jar shown in the figure is not, of course, that described in the text for holding the brain during the process of hardening.

each section measuring from two to two and a half inches in thickness.

After the sections have been carefully examined, and any morbid appearances described and noted, each section is placed in a flat shallow jar (large enough and deep enough to hold it easily) which is then filled up with Müller's fluid; the jar (the edge of which is ground flat for the purpose) is then covered with a glass plate, and the preparation set aside until the following day.

The Müller's fluid should be changed, and the section turned daily, for at least ten days.

In order to prevent any destruction of the section in the process of emptying and turning, I find it advisable to withdraw the fluid by syphon action. After drawing off one syringeful of fluid, the tube (L, Fig. 1) is disconnected from the syringe (care, of course, being taken that while this is being done the end of the tube (L) connected with the syringe is below the level of the jar), and the remainder of the fluid allowed to flow off by syphon action.

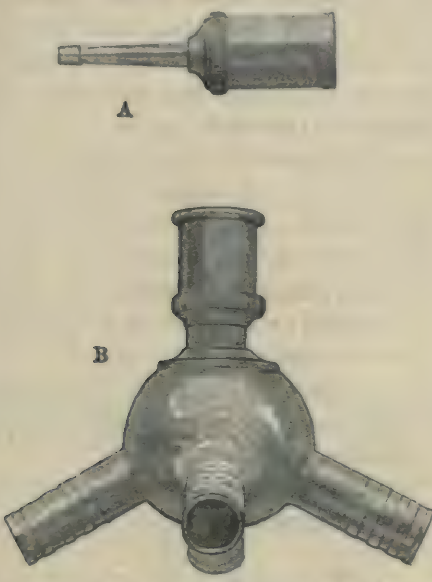


FIG. 2.—CANULA (A), AND DISTRIBUTER (B), NATURAL SIZE.

The brain section is best turned by reversing the empty flat jar, which has, after it is emptied, been covered with a glass plate; the jar is then lifted off the cover, and filled with fresh Müller's fluid, into which the brain section is allowed to glide off the glass plate.

These may seem trifling details, but they are important, inasmuch as they do away with the necessity of fingering the brain tissue, and thereby prevent any possibility of tearing it, while still in the soft, unhardened condition.

At the end of a month or six weeks, these thick portions of brain are sufficiently hard to permit of being cut into thinner sections. The outer surface of sections prepared in this way is considerably shrunken, and any morbid appearances which were present in the fresh state will, in it, be unrecognisable; but the interior of thick portions of brain prepared in this manner is splendidly hardened, permitting of perfectly clean section, and displaying many morbid conditions, such as tumours, softenings, etc., in a very beautiful manner.

I have found this method of hardening in thick sections very convenient and useful; for it allows one to examine the organ while in the fresh condition, and therefore to note changes in vascularity and colour which are destroyed by the process of hardening, while it at the same time enables one to make perfect sections of the whole again.

Professor Hamilton's method of preparing the brain and of cutting entire sections by his large freezing microtome is so complicated and laborious, and requires so much time, as to be entirely unfitted for ordinary every-day use. It is, too, unnecessary for most practical purposes, for which very thin sections are not required; all one wants for most practical purposes is a series of sections which display the naked-eye appearances; the minute changes in any portion of these sections may, if necessary, be subsequently determined in the ordinary (microscopical) manner.

For cutting a series of thin naked-eye sections of the entire brain, the very simple apparatus shown in Figure 3 is all that is required.

It consists of an L-shaped piece of hard wood (A); the horizontal portion of the L (that on which the letter A is placed) measures 11 inches in length, 8 inches in breadth, and 1 inch in thickness; the vertical, short portion of the L measures $7 \times 8 \times 1$ inches.

The piece of brain which is to be cut into sections is placed in the position shown in the figure (see B, Fig. 3), and is firmly pressed against the upright part of the stand by means of a glass plate (C).

The section is then cut by means of a long thin knife, similar to the brain knife used by pathologists, only con-

siderably longer in the blade. The knife is of course held in the right hand, while the left hand presses the glass plate firmly and evenly against the brain tissue. After the section is made, the stand is tilted up, so that the vertical side becomes horizontal; the glass plate is then removed, and the two portions of brain are floated off by immersing the end of the wooden stand, and the brain sections which are lying

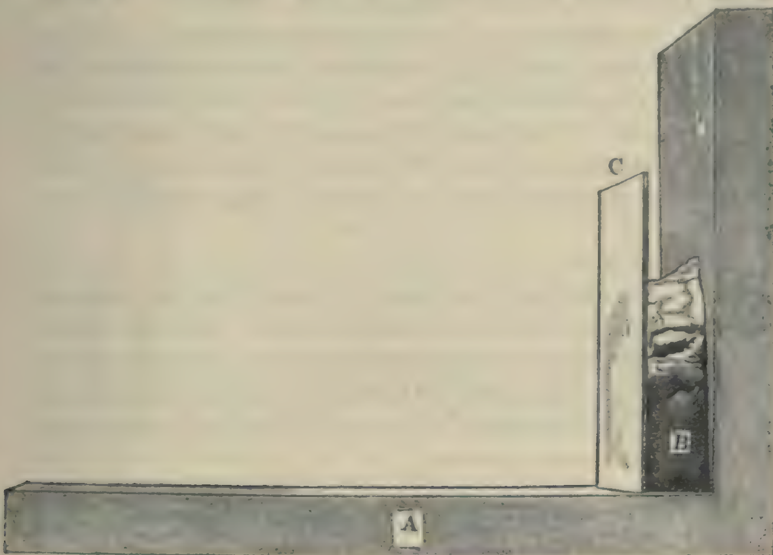


FIG. 8.—APPARATUS DESCRIBED IN THE TEXT FOR CUTTING NAKED-EYE SECTIONS OF THE ENTIRE BRAIN.

The letter A is placed on the horizontal portion of the L-shaped wooden stand; B on the portion of brain tissue; and C above the glass plate by means of which the slice of brain is held in position.

upon it, in water; in this manner all risk of tearing or lacerating the section is avoided.

With a little practice, it is not difficult by means of this simple appliance to cut beautiful, even, sections of a quarter of an inch in thickness.

Sections prepared in this manner can be pinned to a wooden board, and then photographed, should it be thought desirable to preserve their exact appearances.

A series of representations of sections of the entire brain may easily be prepared in this manner; and I have in my

possession a large collection of photographs of naked-eye sections of the entire brain, which it is no exaggeration to say are at least equal to any representations of the normal and pathological brain which have yet been published.

Any portion of these sections may, of course, be cut out, placed in any microtome, cut into thin sections, and examined by any of the ordinary microscopical methods. In this way, a whole transverse vertical section of the brain may, if it is required, be submitted to microscopical examination; with the great advantage, that it is possible to piece as it were the individual microscopical sections together, or at all events to be quite sure of the relative position of any given microscopical section to the entire (transverse-vertical, longitudinal-vertical, or horizontal) section of brain from which it was removed.

Or should it be desired to make thin sections of the entire brain, the naked-eye sections, obtained in the manner described above, may be prepared, frozen, and cut, in the manner recommended by Professor Hamilton. Indeed, my "ready method" is a useful preliminary to Professor Hamilton's method, for the thinness of the sections greatly facilitates and shortens the freezing process.[1]

[1] The apparatus was made for me by Gardner, Forrest Road, Edinburgh.

Clinical Cases.

ON A CASE OF DIFFUSE (SYPHILITIC ?) SCLEROSIS OF THE SPINAL CORD PRODUCING SYMPTOMS OF POSTERO-LATERAL SCLEROSIS.

BY JULIUS DRESCHFELD, M.D., F.R.C.P.

Physician, Royal Infirmary, Manchester; Professor of Pathology, Victoria University.

W. H., æt. 60, porter, was admitted into the Manchester Infirmary on April 7th, 1866.

Family history.—The patient's father died of apoplexy, his mother of dropsy and heart disease. He lost three brothers, one of whom died from consumption; the cause of death of the other two is not known to the patient. One sister is living, and enjoying good health. The patient has three sons and three daughters, all enjoying good health.

Previous history.—He has been employed as a porter in removing furniture; he had to work often twelve to sixteen hours per day for a whole week, and has often exposed himself to cold and wet. He has led a steady life. He has been married twice; the first time forty years ago, the second time eighteen months ago.

Previous health.—He had inflammation of the lungs when a child; 12 years ago he suffered from an attack of gonorrhœa, which, however, soon passed off. He remained well till five weeks after his second marriage, when he noticed a swelling in the left groin, and also some urethral discharge. He did not notice any sore on the penis. He went to his family medical attendant, who told him that he had contracted syphilis. He soon after suffered from an inflammation of the eyes, but had no other symptoms, such as a rash, sore throat, &c. Under active treatment the swelling in the groin disappeared, the discharge ceased, and the inflammation of the eyes subsided. Four months before admission he began to stagger in his walk, and he noticed that he began to lose the control over his legs. Occasionally he felt his legs give way, his legs felt weaker, yet he was able to walk well with the help of stick. He suffered from some pain in the lower part of the spine, but had no pains in the legs, nor did he feel any numbness in the feet or legs. Towards the end of February the patient fell, and soon after noticed that the legs were more paralysed, so that it

was impossible for him to walk. He then also noticed that at times he was unconscious of his passing urine or fæces, while at other times he was conscious of these acts. A few weeks before admission he found that he had lost the control over his bladder completely, and the urine dribbled away. His general health remained fairly good; he never suffered from headache or vertigo.

State on admission.—The patient appears well nourished, but is somewhat anæmic. The examination of the external surface only reveals the presence of a few indurated glands in the left groin and in the back of the neck. The examination of the thoracic and abdominal viscera shows beyond the bladder and rectum affection normal relations. The tongue had a glistening, smooth appearance, and showed the characteristic signs of superficial syphilitic glossitis. The anterior part has quite a lobulated appearance, due to masses of firm fibrous tissue passing in different directions; several small superficial erosions were also visible. The temperature is normal; pulse 88, small, and compressible.

Examination of the nervous system.—The pupils are equal, the left a little irregular in its outline, due to adhesion of the iris. They react but very feebly to light, but better to accommodation. The field of vision is good; the fundus of the eye shows anæmia of the disk, but nothing more. There is no affection of the oculo-motor muscles. There is no affection of any of the other special sense organs or cerebral nerves. Pressure on the parietal bone on either side causes pain. The patient can freely move his head and neck, but is not able to raise himself from the recumbent posture. The cutaneous sensibility of the trunk, both in front and behind, is normal. The spine is not painful on percussion; but the patient complains of spontaneous pain in the lumbar portion of the spine, and also over the epigastrium.

The upper extremities show but few noteworthy changes. They can be freely moved; the muscles show considerable force, and there is no anæsthesia. The movements are co-ordinate as long as the patient keeps the eyes open; but with the eyes shut they become somewhat inco-ordinate, and the finer movements (such as buttoning the shirt) cannot be well executed. The superficial reflexes are normal; the deep reflexes over radius and triceps are present.

The lower extremities are markedly affected. When in the recumbent posture the patient can flex and extend the thigh, can also flex and extend the knee and foot. These movements however are performed slowly, owing to tendency to contractures, and feebly. He cannot raise the whole leg from the bed for more than a few seconds, and abduction and adduction of the whole leg is very feebly performed. The sensibilty to tactile impressions to pain and to temperature is normal. There is however a distinct delay of the perception of tactile impressions on both feet and legs, when compared with the thighs and trunk. With the eyes closed the movements are inco-ordinate, and there is a distinct alteration of the muscular sense. The plantar reflex is present on both sides; the cremaster and abdominal reflexes are absent; the

epigastric reflex is well marked. The deep reflexes are very much exaggerated, and there is well-marked ankle clonus and the phenomenon spinal epilepsy. The electrical reactions of both upper and lower extremities are normal.

The patient cannot hold the urine well; he has to empty the bladder as soon as he has the desire to micturate, and occasionally the urine is passed involuntarily. The urine is alkaline; contain pus s.g. 1012. The bowels are confined.

Diagnosis.—From the history and present symptoms the case was looked upon as a case of syphilitic disease of the spinal cord in the lower dorsal and upper lumbar region, affecting chiefly the posterior and lateral columns. The history of the case and the presence of traces of iritis and glossitis pointed distinctly to a syphilitic affection of the cord, and the onset and symptoms pointed more to an affection of the lateral and posterior column than to a transverse myelites.

Treatment.—The patient was treated with large doses of Iodide of Potassium, and with mercurial inunction.

Progress.—For a few weeks the patient's condition remained stationary, then he improved, and on September 11th we found the following report on his condition:—Patient is now able to move his legs in any direction, he can stand erect, but walking without the help of two sticks is impossible. When he walks the gait is at once spastic and ataxic, there being the stiffness and rigidity seen in spastic paralysis, associated with the staggering gait of ataxia. With the eyes closed the patient falls at once. When in the recumbent posture the patient can move his legs very freely, when he shuts his eyes the movements become very inco-ordinate. The condition of the reflexes is the same as before. The bladder is slightly better, and the patient can hold the water a little longer than before the improvement set in. The bowels are still very much confined. The patient still complains of a pain in the epigastrium and lumbar region.

For the last few days he has been troubled with cough and expectoration; the physical examination of the chest shows extensive bronchial catarrh.

On September 17th the patient had several rigors, and a rise of temperature from normal to 102°. The temperature remained high for a few days, and the urine contained more pus. The chest symptoms improved. During the remainder of September and the whole of October the patient's condition underwent little change. Occasionally there would be a rise of temperature for a few days, ushered in by a rigor and followed by nausea and vomiting. The patient began to lose all control over his bladder, and the urine, in spite of treatment, became highly ammoniacal. The new set of symptoms were attributed to the condition of the urinary organs. The anti-syphilitic treatment was stopped, and the patient put on quinine and salicylic acid. The patient however became gradually worse, the temperature rose high, the appetite left him, he became weaker, though there was a very noteworthy change in the condition of the nervous

system, except that with the general weakness the weakness in the legs also increased. Though the patient could move the legs when in the recumbent posture, these movements were less powerful and extensive. The tendon reflexes remained in the same increased state as before, and the tendency to contractures when any movements were attempted became very marked.

On November 12th the patient had another severe rigor with considerable rise of temperature (104·5), and died on November 15th.

The post-mortem examination was made by Dr. Harris (Pathological Registrar to the Manchester Infirmary). The most noteworthy changes were those found in connection with the urinary organs and the spinal cord. The kidneys were not enlarged, on section they showed several pale and soft spots. The pelvis of the kidney was found dilated and filled with purulent fluid, the ureters were also found dilated; the bladder was found in a state of catarrh, the mucous membrane discoloured and softened, and the contents consisted of purulent fluid.

The brain and its membranes showed no evidence of disease; portions of the cerebral cortex forming part of the motor area were placed in ammonium bichromate, and examined microscopically after having been sufficiently hardened, but showed nothing abnormal.

The several parts of the pons and medulla were carefully examined, and showed no changes.

The membranes of the cord were healthy, and the cord of normal appearance externally, and of normal consistence. Numerous transverse sections of the cord made at different levels showed already to the naked eye certain changes. The transverse sections showed from the upper cervical to the lower dorsal region a patch of degeneration, firmer than the rest of the cord, greyish in appearance, and appearing slightly depressed when compared with the surrounding structure. This patch occupied the region of Goll's tract, and below seemed to extend a little beyond that tract on each side. In the lowest dorsal region the transverse sections had a different appearance. Patches of sclerotic tissue were found occupying nearly the whole of the posterior columns, and extending over the greater part of the lateral columns, and a small patch was found in the anterior columns, corresponding to the region of Turck's tract, a smaller patch on the left, and a larger one on the right side. Successive sections showed that these lesions diminished somewhat in extent and opposite the last dorsal nerve; the sections showed a patch in the posterior columns, extending not quite to the grey matter latterly, and a patch in the lateral columns corresponding to the pyramidal tract. The affected parts in the anterior columns were here reduced to small strips close to the anterior fissure. Sections in the upper part of lumbar cord showed a still further diminution of the sclerotic patch in the posterior columns, and lower still, towards the cauda equina, the only lesion noticed was a pale greyish patch corresponding to the pyramidal tract, whilst the posterior columns appeared perfectly healthy.

The localisation of the lesions in the different sections was particularly well seen in the cord after due hardening in ammonium bichromate.

The microscopic examination of the hardened cord allowed to study more closely the exact extent and the nature of the affection. For this purpose the sections were stained both in carmine and after Weigert's method.

Throughout the whole of the cervical and upper dorsal region the affection was limited to Goll's tract, reaching posteriorly to the pia mater, which in itself was not altered, and reaching anteriorly to close but not quite up to the posterior commissure. In the lower dorsal region the lesion extended latterly to the adjacent parts of Burdach's tract, but did not reach the posterior horn, while anteriorly it extended quite up to the posterior commissure in the centre, but not at the sides.

The anterior and posterior horns appeared perfectly normal.

For the lower dorsal region the affection was of the nature of a diffuse sclerosis, and included Goll's tract and Burdach's tract, with the exception of a small strip on the inner boundary of the posterior horns; the lateral columns were affected almost in their entirety posteriorly, anteriorly the affection stopped short a little of the anterior horns, externally it reached as far as the pia mater, which here again appeared unaffected.

In the anterior columns the affection seemed limited on the right side to Türck's columns, while on the left side only a small strip, adjacent to the anterior fissure, showed a sclerotic condition. The anterior and post. grey matter showed some cell infiltration, the arteries here showed well-marked endarteritis, and by means of Weigert's method changes in the ganglia cells and nerve fibres in Clark's columns were made evident.

In the lowest dorsal and upper lumbar region the affection corresponded closely as regards extent to a postero-lateral sclerosis. In the posterior columns Goll's and Burdach's tracts were affected; but it was noted that the affection did not extend beyond a line drawn vertically from the innermost part of the substantia gelatinosa to the posterior periphery, whilst the changes in the lateral columns seemed entirely limited to the region of the pyramidal tract. The affection in the anterior column was only apparent on the right side, in the form of a small stripe close to the anterior fissures, the left anterior column appeared normal. The grey matter was affected similarly as in the lower dorsal region, as stated above.

For the lower lumbar region the affection was entirely limited to a small triangular patch on the outer and posterior part of the lateral columns, whilst the posterior columns, the anterior column and the grey matter appeared fairly healthy, with the exception of slight cellular infiltration and enteritis.

The pia mater, the posterior and interior roots appeared healthy throughout the length of the cord.

As regards the histological changes, we have to notice numerous blood-vessels with thickened walls, the thickness depending in

part on an endarteritis, which in some places caused marked diminution of the lumen of the vessels, and in part on an infiltration of the perivascular sheath. Round the vessels were noticed masses of round and spindle cells, arranged in some spots concentrically round the blood-vessel. The rest of the altered tissue showed numerous nuclei, tracts of fibrous tissue, thick and coarse, fine granular matter, a sparing number of Deiters' cells. The nerve fibres showed the usual changes, hypertrophied axis-cylinder nerve fibres, in which the white substance had taken the carmine-staining and atrophied nerve fibres. In the grey matter the blood-vessels showed likewise endarteritic changes and perivascular infiltration. Besides these a large number of nuclei and fine granules were seen in the neighbourhood of the ganglia cells. The cells themselves (with the exception of those in Clarke's columns) showed but little alteration; some of the cells in the anterior horns however had a hyaline appearance, in others the nuclei had not taken the staining, and others had their cell processes broken or shrunk.

The case presents several points of interest.

The first question which arises refers to the nature of the affection of the cord. Have we here a syphilitic imflammation, or is the sclerosis (for such the naked eye and microscopic appearance declare it to be), though etiologically connected with syphilis, yet not itself of a syphilitic nature? Does it stand in the same relation to the syphilis as locomotor ataxy coming on in a syphilitic subject? Though the question cannot be answered positively, yet there are several factors which point to the lesion as of syphilitic nature. Thus the symptoms came on soon after the syphilitic affection, whilst in most cases of locomotor ataxy syphilis has preceded the ataxic symptoms by a much longer period of time; the symptoms also yielded at first to an anti-syphilitic treatment, whilst such a treatment has no effect in the largest majority of cases of locomotor ataxy.

The anatomical structure likewise presents some peculiarities speaking in favour of syphilis. Thus in a non-syphilitic affection we should expect at so early a period after the onset (for the disease lasted scarcely twelve months in our patient) to find an appearance corresponding more to myelites than to sclerosis. The histological examination showed extensive endarteritis and penarteritis, and though this can no longer be considered as characteristic of syphilis, yet it has, as regards the nervous system, been found most markedly in syphilitic sclerosis. Thus Rumpf[1] gives a case of diffuse syphilitic infiltration of the cord where the histological

[1] 'Die Syphilitischen Erkrankungen des Nervensystems,' p. 341, and 'Arch. f. Psychiatrie,' vol. xvi. p. 410

changes were very similar to those found in our case; Greiff[1] describes a case of syphilis with paresis of the lower extremities where in the vessels well-marked obliterating endarteritis was found. Ballet and Minor[2] describe a case of diffuse sclerosis in a syphilitic subject which, both as regards the histological changes and in many respects also as regards the localisation, resembles our case, and draw special attention to the vascular changes which cause them to distinguish between a perivascular sclerosis and a peritubular sclerosis (seen in the ordinary primary inflammation of the cord, and for which the term parenchymatous inflammation would perhaps be better). Savard[3] likewise draws attention to the vascular changes, and to the cell infiltration and poliferation in syphilitic diseases of the cord. I could besides cite Charcot and Gombault,[4] Homolle,[5] Leyden,[6] Westphal,[7] and Schulze.[8] We have however to bear in mind, that similar changes are found in the non-syphilitic form of sclerosis, especially in acute and chronic myelitis, and I have seen repeatedly very marked endarteritis in otherwise healthy cords of persons who have died from granular kidney; as was long ago pointed out by Gull and Sutton. In many of the recorded cases of syphilitic sclerosis the inflammatory process affected the pia mater also; this, however, was not so in our cases, where the pia mater, as already stated, was perfectly intact.

Another feature of the lesion was its diffuse character; it affected not only the white matter, but also, though to a much smaller extent, the grey matter of the cord. This has also been found in some of the cases of syphilitic myelitis, especially in the case of Rumpf cited above; yet such a diffuse infiltration occurs also in the ordinary non-specific form of myelitis.

From these considerations it is evident that, though the syphilitic nature of this infiltration is highly probable, yet we cannot speak on this point very positively. The detection of the syphilis bacillus might in these cases materially assist us in our differential diagnosis, but as yet our methods of staining these bacilli cannot be said to be satisfactory, and as they are but sparingly found even in primary syphilitic sores, it is not very likely that our efforts to detect them in syphilitic

[1] 'Arch. f. Psych.,' vol. xii. p. 564.
[2] 'Arch. de Neurologie,' vol. vii. p. 44.
[3] 'Etude sur les myelites syphilitiques,' 'Thèse de Paris,' 1882.
[4] 'Arch. de Phys.,' 1873, p. 143.
[5] 'Progrès Méd.,' 1876.
[6] 'Charite Annalen,' 1876, p. 260.
[7] 'Arch. f. Psychiatrie,' vol. vi. p. 245.
[8] Ibid., vol. viii. p. 222.

infiltrations of the cord would be very successful. Unfortunately the cord from our case had been kept in ammonium bichromate for some time, which would render an examination for the bacillus still more difficult, if not altogether impossible, for it is doubtful whether the method by means of which we can detect the tubercle bacillus in tissues hardened in ammonium bichromate is also applicable for the bacillus found in syphilitic lesions.

Our case presents another point of interest. The affection in the posterior columns, especially in the upper lumbar region, did not extend beyond a line drawn vertically from the substantia gelatinosa to the posterior border of the cord; the case thus supports Westphal's observations on the persistence of the knee tendon reflex in posterior sclerosis in such cases, and we can thus understand its presence in our case, and its exaggeration owing to the implication of the lateral columns.

If we sum up our case briefly, we have a patient who soon after having been affected with syphilis shows symptoms of ataxic paraplegia with affection of the bladder, and as the cause of this we find a diffuse sclerosis of the lower dorsal region of the spinal cord affecting for a short extent the anterior lateral and posterior columns, and limiting itself in the lowest dorsal and upper lumbar region to the Goll's and Burdach's tracts in the posterior columns, the indirect pyramidal and direct cerebellar tract (as far as they extend) in the lateral columns, and the cells and fibres in Clarke's columns.

The changes in the upper dorsal and cervical region we are inclined to look upon as secondary ascending, those in the pyramidal tract in the lower lumbar regions as secondary descending degeneration. The histological examination of these parts showed a somewhat different appearance when compared with that in the lower dorsal region: the vascular and perivascular changes were much less marked, the neuroglia showed a larger quantity of granules, but fewer nuclei, and many of the axis-cylinders in the affected zone had still a normal appearance.

Cases like the one given are by no means rare, but they have been mostly described as postero-lateral sclerosis, and have been looked upon as combination of several systemic affections, *i.e.* of Goll's tract with the adjacent portions of Burdach's tract, the pyramidal tract, and cerebellar tract.

That such combined affections do exist can no longer be doubted. The cases related by Strumpell,[1] Westphal,[2] Kahler,

[1] 'Arch. f. Psych.,' vol. xi. p. 27, and vol. xvii. p. 217.
[2] Ibid., vol. xv. p. 224.

and Pick,[1] and more recently by Babinsky,[2] and several others, show this; such cases are, however, by no means common. Clinically they are chiefly characterised either by spastic paraplegia, without implication of the bladder and rectum, and without many sensory disturbances, like one of the cases recorded by Strumpell, or we have symptoms characteristic of tabes dorsalis (*i.e.* inco-ordination, absence of tendon reflexes, and sensory disturbances, etc.), together with marked paralysis and contractures.

A number of other cases, however, have been classed by some authors, such as Grasset,[3] who has collected no less than thirty-three fatal cases, under the heading of Combined Tabes, which included many cases like ours, of diffuse sclerosis.

We are inclined to agree with Ballet and Minor,[4] who speak of these cases as false combined systemic sclerosis. A glance at the post-mortem appearances, as described by some of the authors, shows at once that the lesions found are by no means always the same. The posterior columns are affected in a very varying degree, both as regards lateral and vertical extension; in some cases, like those of Zacher[5] and Déjérine,[6] and both Goll's and Burdach's tracts were affected, at least in some regions of the spinal cord, whilst in others (Strumpell, Westphal) the lesion in the post. columns was almost entirely limited to Goll's tracts. The pyramidal tract also showed by no means the uniform well-defined lesion as seen f. i. in amyotrophic lateral sclerosis or secondary descending sclerosis; the direct pyramidal tract in the anterior columns has but rarely been found affected, and the cerebellar tracts, while distinctly affected in some cases (Strumpell, Kahler and Pick, Westphal), were found intact in others. In some cases moreover the pia mater showed signs of inflammation.

With such a variability of the lesions, it is not astonishing that the symptoms of the so-called postero-lateral sclerosis should be so diverse.

In some cases[7] we have the symptoms of locomotor ataxy, to which are added more or less pronounced paralysis and contractures; in others (like those of Strumpell cited above) we have almost pure spastic spinal paralysis, with scarcely any sensory troubles, and with increased tendon reflexes; in some, bladder and rectum are affected early in the disease, in others these

[1] 'Arch. f. Psych.,' vol. viii. p. 251, etc.
[2] 'Revue de Médecine,' 1887.
[3] 'Arch. de Neurolog.,' vols. xi. and xii.
[4] Ibid., vol. vii. p. 44.
[5] 'Arch. f. Psych., vol. xv. p. 405.
[6] 'Arch. de Phys.,' 1884, iv.
[7] Kahler and Pick, 'Arch. f. Psych,' vol. viii. p. 252; Westphal, 'Arch. f. Psych.,' vol. viii. p. 470.

organs may remain unaffected, or become only slightly affected towards the end.

We can thus no longer speak of one form of combined postero-lateral sclerosis, but must admit, both from the clinical and pathological standpoint, several forms; those resembling tabes dorsalis and those resembling Friedreich's disease,[1] and those resembling primary lateral sclerosis.[2]

There remain however still a large number of other cases, which, like the one given above, are due to a diffuse sclerosis. That the symptoms in these cases should vary in the individual cases is easily understood, and be dependent on the situation and extent of the sclerosis in the posterior and lateral columns respectively. These cases then, clinically considered, are difficult to distinguish from cases of pure combined systemic sclerosis except by their history and course, such as the gradually developing paresis with contractures and the presence of bladder and rectum troubles. In many cases the upper extremities remain free, and show neither ataxic nor paretic symptoms.

Pathologically the chief distinction lies in the vascular and perivascular changes, and the marked increase of the fibrous element in the neuroglia, whilst the nerve-fibres show still a fair number of normal or simply atrophied axis-cylinders. The process in some cases can be traced from the pia mater, which is found thickened and altered by inflammatory deposits.

[1] Kahler and Pick; Schulze.
[2] Strümpell.

HYDATIDS OF THE SPINAL CANAL.

BY ROBERT MAGUIRE, M.D.

Physician to Out-Patients, and Joint Lecturer on Pathology, St. Mary's Hospital.

THE spinal canal is one of the rarest of localities for the development of hydatids. Therefore, while reporting a case of this kind which occurred at St. Mary's Hospital, I have collected for comparison the whole of the cases on record, twenty in number, together with two instances in which cysticerci were present in the spinal cord, in order that the symptoms and pathological signs may be compared, and in this way the natural history of the disease made evident. The new case I have to report was under the care of my colleague, Dr. Lees, who has kindly placed it at my disposal, and the records of the case were furnished by P. A. Lloyd, F.R.C.S., the house-physician in charge of the case, who carefully observed it throughout.

F. L., aged 16, was admitted into St. Mary's Hospital, on the 5th of July, 1886, complaining of weakness in his legs, and inability to walk. For the last nine months the patient had worked in a bakehouse, and six weeks previous to admission had frequently bathed when perspiring freely, sometimes remaining in the water for an hour and a half. He attributed his illness to a chill obtained in this way.

Four years ago the patient had scarlet fever, and three years ago he attended as an out-patient for paresis of the left forearm. He then had sharp pains in the arm, chiefly at night, but these passed off when at his work. The loss of power in the forearm disappeared under the influence of galvanism. Nothing of importance was elicited as to the family history.

The present illness began about two months before admission with a sharp pain across the upper part of the back extending from shoulder to shoulder. The pain increased, and on the 24th of June, he first noticed weakness in the legs; about a week before admission he was treated by a medical man for rheumatism.

On admission, the patient had pain in the region of the upper dorsal vertebræ; he could walk alone without dragging either foot, but his gait was unsteady and awkward. The feet were thrown forward and brought down suddenly, the heels striking

the ground first. There was no tottering when he stood with his feet together and his eyes shut. There was no muscular atrophy. The electrical reactions of the arms were normal; in the legs there was excessive irritability to the faradic current. The limbs were not generally rigid, but marked tension and rigidity of the ham-string muscles was observed when the patient sat up. The knee-jerks were excessive, and ankle-clonus could be obtained on the right, but not on the left side. Plantar reflex was marked, abdominal and cremasteric reflex could not be obtained, and the epigastric reflex was slight on the left side, absent on the right. There was perfect control of the sphincters, but a history was obtained of incontinence of urine during the previous week. The pupils were equal, and nothing abnormal was seen in the fundus of either eye. Sensation was normal, there was no tenderness over the spine and no external evidence of caries of the vertebræ. There were no cerebral symptoms.

The thoracic and abdominal organs appeared healthy. The urine was very feebly acid, almost neutral, sp. gr. 1028, and deposited triple phosphates, but no casts or renal epithelium. The temperature was normal.

Three days later (July 8th) the patient could not walk without assistance, and the gait was of a shuffling character. He now fell, on attempting to stand, with his eyes shut and feet together. Flexion and extension of the legs and thighs could be performed fairly well when the patient lay in bed; he could turn on his face or get out of bed without help, but all the movements were done slowly and with effort. The arms were not affected. The pupils at times appeared unequal, the left being contracted and the right of ordinary size, but both responded well to light and to accommodation.

On the 14th of July, the patient had less power in his legs, and for the first time since admission passed urine and fæces involuntarily. The pupils were now normal in size and reaction. The patient could not stand alone. Ankle-clonus was on this day marked on the left side, and could just be obtained on the right. The knee-jerks were somewhat more powerful than on admission.

On the 20th of July the patient could not move his legs without assistance, but on tickling the feet partial flexion of the knee was obtained. Ankle-clonus was marked on the left side and was also present on the right. The sensation of the legs appeared to be defective, but the patient's statements were inconsistent. The pain in the back had ceased, and complaint was made of numbness below the knees. About this time severe pain was experienced in both elbows, although no swelling, redness, or tenderness could be there discovered. The urine was now alkaline and was passed involuntarily. The bowels were constipated. The power in both arms became less, the right hand gauging 25 and the left 20 degrees of the dynamometer.

On the 30th of July, the pupils were very contracted, and only slight dilatation was produced by shading them from the light. There was great tension of the muscles of the neck and retraction

of the head. Severe pain in the head was complained of. The pain in the elbows disappeared, but the arms became almost totally paralysed. The patient could not now move the legs at all, and had no control over micturition or defecation. There was no bed-sore. On the 29th, the temperature, which had remained normal throughout the illness, rose to 102·6° F., but fell next day to normal, and in the further progress of the case never rose above 100° F.

On the 3rd of August, the patient had two severe attacks of dyspnœa, the lungs showed signs of congestion and œdema, and slight epistaxis occurred during the night. Death took place on the 5th of August from asphyxia, the patient remaining conscious to the last.

The treatment during the patient's stay in hospital consisted of counter-irritation by flying blisters applied to the spine, and the internal administration of bromide of potassium and ergot.

The autopsy was made twelve hours after death. The membranes of the brain were congested and the cerebral fluid was excessive in amount. The brain itself was normal. On removing the vertebral arches to expose the spinal cord, a large number of cysts were found lying between the dura-mater and the last cervical and upper six dorsal vertebræ. The cysts varied in size from that of a pin's head to that of a small chestnut. Some were shrivelled and contained an opalescent fluid. The fluid from most of the cysts was clearer, and showed under the microscope hooklets and echinococci. Some of the cysts were imbedded in the bone of the vertebral arches, others pressed upon the spinal cord. The latter was so extremely soft and congested in the region of the cysts and immediately above and below this part, that no further examination could be made. No cysts were found in any other part of the spinal canal or in any other organ of the body. The lungs were œdematous, and pleuritic adhesions were found on the right side. The liver and kidneys were congested, but otherwise normal.

The cases similar to this which I have been able to find on record are the following:

1. Chaussier.[1]—A woman, aged 22 years, suffered from paralysis and loss of sensation in the lower limbs. She was pregnant, and spontaneous accouchement occurred without pain. Four days afterwards, fever and suppression of lochia set in, and death ensued upon the tenth day, five or six months after the first spinal symptoms. A large hydatid cyst was found in the thorax, and the third and fourth dorsal vertebræ were eroded in many places. Within the vertebral canal were found about a dozen acephalocysts

[1] 'Jour. de Méd. de Corvisart,' vol. xiv. p. 231. 1807.

compressing the spinal cord, in the region of the first four dorsal vertebræ.

2. Esquirol.[1]—A woman, aged 53, began to suffer from convulsions. The attacks became more frequent, and the patient died in status epilepticus, after five days of coma, at the age of 56. Acephalocysts were found in the arachnoid cavity, extending from the medulla to the end of the lumbar region. The cord was softened in the lower part. A cyst containing brown fluid was found in the pituitary body.

3. Reydellet.[2]—A woman, aged 22 years, suffered from pain in the shoulders and right arm, with weakness of the arm. In three years' time there was no pain, but only weakness. Still later, pain in the spine was experienced. Then appeared anæsthesia of the legs, and paralysis of the right leg. A tumour was now found in the lumbar region, and pressure upon the tumour increased the pain in the arm. The tumour was opened, and a large number of hydatids removed from it, and from the vertebral canal, with which the interior of the tumour communicated. The patient improved for a time, but abundant suppuration set in and complete paraplegia appeared. Death, however, did not occur until more than a year after the opening of the tumour.

4. Morgagni[3] describes a case in which hydatids perforated from the outside into the spinal canal. A woman, aged 26, was confined during the last six months of 1814. Ten months later she began to complain of pain in the lumbar region, lancinating in character, but not sufficiently intense to greatly incommode her. As the disease progressed, formication, cramps and numbness in the lower extremities were complained of, and soon sensation and motion became affected. The patient finally became completely paraplegic, urine and fæces being passed involuntarily. The patient died nine months after the first onset of symptoms. At the autopsy there was found beneath the peritoneum, near the left kidney, an elastic tumour, round, and about the size of a fist, which had eroded the bodies of the first and second lumbar vertebræ. It contained a large number of hydatid cysts. The intervertebral foramina were greatly enlarged, and through them the acephalocysts gained access to the spinal canal. The duramater of the cord was surrounded by hydatid cysts of all sizes, and the lumbar nerves were compressed.

5. Mélier.[4]—A woman, 29 years of age, complained of pains, spasms, paralysis and loss of motion in the legs for three years. After death a small cyst was found in the soft parts of the dorsal

[1] 'Bulletin de la Faculté et de la Société de Médecine de Paris, t. v. p. 426. Obs. vii. 1817.

[2] 'Dict. des Sciences Medicales,' art. Moelle, t. xxxiii. p. 564. 1819.

[3] 'De Sedibus et Causis Morborum,' vol. v. p. 168. 1822.

[4] 'Journ. gen. de med.' Sedillot, 1825, p. 33.

region filled with acephalocysts. On tracing this it was found that it had eroded the laminæ of the fifth and sixth dorsal vertebræ, and in the vertebral canal, between the dura-mater and the bone, numerous cysts were seen. The vertebral canal was enlarged at this spot from erosion of the bones, and the spinal cord compressed and softened.

6. MONTANSEY[1] reports the case of an idiotic and epileptic woman. At the autopsy there was found a large number of hydatids on the surface, and in the thickness of the brain and cerebellum, and "a score in the thickness of the spinal cord."

7. MAZET.[2]—A man presented himself at the Hôpital St. Antoine at Paris, with an abscess situated over the posterior superior spine of the ileum. The abscess was punctured and a quantity of excessively fetid pus liberated. The patient died, and the end of the vertebral canal was found filled with acephalocysts. The sacrum was carious, and its canal communicated with the abscess by an opening situated in the posterior median line. There was no paralysis during life.

8. DUMOULIN.[3]—A man, aged 25 years, eighteen months before his death, suffered from pain in the back after a blow. The pain increased in severity. The legs became weak, the movements difficult, and the power of sensation diminished in the lower limbs. The bladder and rectum were paralysed. One month before death, complete paraplegia set in. At the autopsy a cyst was found between the muscles of the back and the vertebræ, and the vertebral laminæ were much thinned. Twelve hydatid cysts were found free in the vert bral canal, lying between the dura-mater and the second and fifth dorsal vertebræ.

9. DUBOIS.[4]—A girl, aged 20, suffered from pain in the lumbar region for one year before death. There was also weakness of the limbs. Pinching the legs caused pain in the soles. Signs of caries of the second dorsal vertebra were seen, but there was no tumour and no abscess. Bed-sores formed, the urine and fæces were discharged involuntarily, and death occurred. Hydatid cysts were found in the eleventh and twelfth dorsal vertebræ, which had penetrated the vertebral canal and compressed and softened the cord. No other hydatids were found.

10. CRUVEILHIER[5] records the case of a woman, aged 38, who was seized with complete paralysis of the lower extremities. There were sharp continuous pains in the lower extremities, and there was a burning feeling in the same parts. The slightest

[1] 'Bulletin de la Soc. Anatom.' 1827, p. 188.
[2] Ibid. 1837, p. 2?6.
[3] Ibid. 1847, p. 321.
[4] Ibid. 1848, p. 95.
[5] Ibid. 1850, p. 63; and 'Anat. Pathol.' xxxv., pl. vi.

passive movement of the lower limbs caused pain. Simple touch caused pain which ran from the point touched throughout the length of the limb. There was no plantar reflex. At the level of the twelfth dorsal, or first lumbar vertebra, there was discovered a soft point easily depressed, becoming prominent on coughing, which was believed to be an uncured spina-bifida. At the autopsy, in the situation of this soft spot was found a pouch filled with acephalocysts. The spines and laminæ of the vertebræ were eroded and the cord pressed upon, although the dura-mater was unchanged. The spinal cord beneath the cyst was much softened, and changed into a purulent sac, so that at the point of compression scarcely a trace of spinal cord remained.

Cruveilhier states that he has seen several cases of the kind, but gives no further details.

11. Goupil.[1]—A man, aged 40, had suffered for some time from weakness of the legs. On the 15th of May he had walked from Montmartre to the Hôpital Beaujon, but on the 17th could not walk at all. Sensation disappeared from the legs. A bed-sore formed on the sacrum, and the patient died twenty-one days after entering the hospital. At the autopsy, an acephalocyst was found in the lumbar region of the spinal canal at the posterior part of the cord outside the dura-mater. The spinal cord was softened at the point in contact with the cyst, and the bones were not affected.

12. Ogle.[2]—A case occurred at St. George's Hospital, in which a cyst containing a vast number of small ones was found within the substance of the spinous process of the seventh cervical vertebra, projecting upon the spinal cord and producing symptoms not unlike those of cancer. No details are given of the clinical history.

13. Foerster[3] mentions a case in which echinococci had produced caries of the vertebræ, abscess between the muscles of the back, perforation of the dura-mater, and purulent inflammation of spinal cord and its membrane.

14. Bartels.[4]—Man, aged 25, complained of pain in the left arm, four months before admission into hospital. Afterwards, pain in right arm and neck came on, with gradually developing symptoms of compression paraplegia, which proved fatal some three months after admission. At the autopsy, the dura-mater spinalis was very congested. On section, clear fluid flowed away. There were no changes in the vertebræ. Immediately below the cervical enlargement of the cord was a cyst, lying between the dura-mater and the cord, compressing the latter from behind forwards, and towards the right side. The length of the depression in the cord was 3·5 cm.,

[1] 'Bulletin de la Soc. Anatom.' 1852, p. 211.
[2] 'Pathol. Trans.' xi, p. 299.
[3] 'Handb. d. Pathol. Anat.' vol. ii, p. 639.
[4] 'Deutsches Archiv f. Klinische Medizin,' vol. v, 1869, p. 108.

while the cyst itself, after being opened, measured 5 cm. in length. At a distance $7\frac{1}{2}$ cm. below the first was another similar cyst, also on the left side of the cord, and within the dura-mater, measuring 3·8 cm. in length. Above the first cyst, the spinal cord was normal; where compressed by the cyst, it was pale and soft. Between the two cysts again the cord was fairly normal, but, below the second cyst it was much softened, and the pia-mater congested. No other cysts were found in the body.

15. ROSENTHAL.[1]—A patient, aged 15 years, began to suffer, without any apparent cause, from pain and weakness, in both lower extremities. In three months he could not leave his bed. There was observed cramp of the flexors, followed by that of the extensors, with twitches and formications, and afterwards total paralysis. Total anæsthesia was found in the lower extremities as high as the nipples, and backwards to a line through the scapulæ. Later on, incontinence of urine and fæces appeared, with bed-sore; and death occurred from pyæmia. At the autopsy, the brain was pale, the right pleural cavity contained turbid fluid, and the right lung was compressed. Between the pleura and the bodies of the third to the fifth dorsal vertebræ, there was a sac the size of a goose egg; this had eroded the bodies of the vertebræ, and entered the spinal canal on the right side. It lay outside the dura-mater, and compressed the cord between the third and fourth dorsal nerves, until only a thin layer of nerve-substance was left.

16. MURCHISON[2] describes a specimen existing in the Middlesex Hospital Museum, in which two hydatid cysts, one on each side, separated the pleura from the ribs and the sides of the bodies of the vertebræ. The hydatids were opened in sawing through the laminæ of the vertebræ, and the spinal cord at this spot was considerably smaller than elsewhere. The patient was a woman, aged 40, who had been admitted into the hospital with paraplegia, and retention of urine. She died with a large slough on the sacrum, and the bladder was found to be inflamed. There was a large hydatid cyst in the liver.

17. LIOUVILLE AND STRAUSS.[3]—A man, aged 42, had paralytic symptoms in the lower limbs for six months before death. A hydatid cyst was found in the back communicating with débris under the scapula, and running down under the muscles of the back. The cyst entered the spinal canal between the ninth and tenth ribs, disorganising the laminæ of the vertebræ. In the spinal canal, hydatid cysts were found, ranging in size from that of a pea to that of a nut, lying outside the dura-mater, and compressing the cord. The dura-mater was adherent at several points to the hollowed vertebral bodies. Sudden paraplegia was the first

[1] 'Handb. d. Nervenkrankh.,' pp. 192–193.
[2] 'Diseases of the Liver,' 2nd ed. p. 129.
[3] 'Bulletin de la Soc. Anat.' 1875, p. 93.

symptom. Beyond the point where the cord was affected by the cyst, descending and ascending changes were seen.

18. LLEWELLIN.[1]—A patient under the care of Dr. Annand, of Australia, had a hydatid cyst, which occupied the whole length of the vertebral canal. The cyst was tapped during life, and fluid containing echinococcus hooklets was removed.

19. WOOD, H. S.[2]—A woman, aged 54, was seized on the 11th of February with darting pains in the limbs, and on the following day had incontinence of urine. On February 20th these symptoms had disappeared, but on March 28th she was re-admitted to hospital, suffering from incontinence of urine, a feeling of numbness of the left arm and leg—the movement of the leg being impaired, but sensation perfect. On the 6th of April the right arm and leg became powerless, and the patient died comatose on the 7th of April. On post-mortem examination, there was found opposite the last lumbar and upper sacral vertebræ a hydatid cyst within the dura-mater of the cord, filling the spinal canal, and extending through the second left anterior sacral foramen, so as to form a small bilocular cyst on the posterior wall of the pelvis, covering the points of exit of the second and third sacral nerves. The cyst also extended through the first and second sacral foramina, which were partially thrown into one. The whole cyst and its prolongations were full of small daughter cysts. At the upper end the cyst was open, and some daughter cysts were lying free. There was an immense quantity of fluid effused beneath the membranes of the cord and brain. The liver also contained a hydatid cyst.

20. MOXON.[3]—A woman, aged 58, had suffered for eleven months from pain in the side, and had been paraplegic for six weeks. A multi-locular hydatid was found budding externally; it formed a large elastic swelling on the left side of the spine, and made its way into the canal through the second and third lumbar vertebræ. It compressed the cord, but did not penetrate the dura-mater.

DIXON records a hydatid cyst of the neck, which had eroded the bodies of the fifth, sixth and seventh, cervical, and of the second dorsal vertebræ. It was opened, and the patient died of hæmorrhage, but it is not clear from the record whether the cyst entered the spinal canal or not.

In the following two cases cysticerci were found in the spinal cord.

21. WESTPHAL[5] describes a case in which the symptoms were mainly those of epilepsy. Cysticerci were found in the brain.

[1] Quoted by Cobbold, 'On Parasites,' p. 140.
[2] 'Austral. Med. Journ.' 1879, p. 222.
[3] Quoted in Fagge's 'Medicine,' vol. i. p. 413.
[4] 'Med. Chir. Trans.' vol. xxxiv. p. 315
[5] 'Berlin Klin. Wochenschr.' 1865, p. 425

At the lower part of the spinal canal, in the region of the cauda equina, was found a large number of small cysts containing fluid and no special solid elements. Some of the cysts were attached to one another. The spinal cord was somewhat softened to the naked eye, but otherwise unchanged.

22. WALTON[1] describes a case which occurred in Leipsic, under Wagner. A butcher's widow, aged 56, was admitted to hospital with complete anaesthesia and almost complete paralysis of the legs. There was also observed a loose condition of the knee and ankle joints. There was no reflex action in the legs, and the urine and fæces were passed involuntarily. The spine was normal. There was muttering delirium and fever, the temperature ranging between 37° C. and 41° C. At the autopsy, the brain showed opacity of the arachnoid and several cysticercus cysts on the surface and in the anterior part of the right corpus striatum. In the cord was found sclerosis of the posterior columns, as in tabes. The grey matter of the posterior horns was affected, but the anterior horns were normal. The left side of the cord at the level of the third and fourth spinal nerves was larger than the right, and here a cyst was found in the grey matter of the left side. The cyst was 6 mm. × 5 mm. in size, and had caused atrophy of the ganglion cells in its neighbourhood. It was surrounded by firm connective tissue. Inside the cyst was found a cysticercus head with hooklets.

Rokitansky gives no case, but states that cysticerci sometimes appear in the spinal cord.

The new case I have reported requires but little comment. The paresis of the left arm which occurred some time before the symptoms of the last illness set in, was probably unconnected in any way with the hydatid tumour. The actual symptoms of the disease were clearly those of pressure upon the spinal cord below the origin of the arm nerves, causing the paralysis and irritative phenomena of the lower limbs. Consequent upon the pressure, myelitis resulted which produced the arm symptoms and probably caused the death. The retraction of the head and tension of the muscles of the neck may be noted, although I consider them to be merely accidental symptoms. Nevertheless, Ollivier,[2] in commenting upon Chaussier's case mentioned above, lays special stress upon the rigidity of the neck; he remarks that it is due to a permanent irritation by the parasites.

In only four cases, namely, No. 2 (Esquirol), No. 6 (Montansey), No. 14 (Bartels), and No. 19 (Wood), had the hydatids developed inside the dura-mater, whereas, in both

[1] 'Bost. Med. & Surg. Journ.' 1881, p. 511.
[2] 'Traité de la Moelle Épinière,' vol. ii. p. 527.

the cases of cysticercus reported, the parasite was found in the substance of the spinal cord or its prolongation. In some of the other cases, in which the cysts were found between the dura-mater and the bones, it is clear that the growth had commenced within the vertebral canal, or at least in the bones which bound the canal. The records of two of these cases, namely, No. 11 (Goupil) and No. 18 (Llewellyn), show that the bones were not affected by the growth, proving that it is possible for the parasite to develope in the soft tissues of the canal. In the other cases, however, definite affection of the bones is described. They may have been eroded by the growth of the cyst, but bearing in mind the frequency with which hydatids are found in the bones of other parts, it would appear probable that here also their primary seat was the vertebral column. In the original case described in this paper, the cysts were actually imbedded in the spinous processes. In seven of the cases described it will be seen that the vertebral canal was only secondarily invaded; while in three: No. 1 (Chaussier), No. 3 (Reydellet), and No. 5 (Mélier), the record leaves the exact origin of the cyst in doubt. It will be noted in several of the cases quoted, and also in the new case described, that numerous cysts were found. It is most likely that these were really daughter cysts, the wall of the mother cyst having disappeared under the pressure. Such is the comment made by Foerster,[1] with reference to his own case.

The symptoms of the affection are those of compression paraplegia, and, unfortunately, in the absence of some external sign of hydatid tumour, there appears no means of diagnosing the cause of such compression. This is the more to be regretted, as a consideration of the cases given shows that, in by far the majority of cases, the cyst is situated at the posterior surface of the cord, and therefore would lend itself readily to operative interference. In one case, No. 18 (Llewellyn), the position of the cyst is not fully described, and in four cases, as already mentioned, the cyst was in the substance of the cord; but of the remaining sixteen cases, there are only four, Nos. 1, 4, 15 and 16, in which the cord was affected from the anterior part of the canal. Finally, it will be observed that in Reydellet's case an operation was performed, and even at this early date (1819), the vertebral canal was opened and cleared of its hydatid tumours. The operation was so far successful that the patient lived for twelve months and then died of suppuration. Modern surgery would probably avoid such a termination.

[1] *Loc. cit.*

SOME FURTHER OBSERVATIONS ON FRIEDREICH'S DISEASE.

BY J. A. ORMEROD, M.D. (OXON.), F.R.C.P.

It had been my intention in this paper to continue the digest of cases of Friedreich's disease which I published in a former number of 'Brain.'[1] But seeing that Dr. Judson Bury has recently, in his valuable article,[2] dealt exhaustively with the literature of the subject, I shall confine myself to recording some further cases which I have observed, and adding references to some papers which have appeared since Dr. Bury's publication.

My first set of cases are from a family resident near Hertford, comprising—Case I. A young man in whom the principal symptom has been progressive ataxia of the lower limbs. Case II. An elder sister, exhibiting the typical symptoms of advanced Friedreich's disease, plus sundry attacks of vomiting and of a syncopal (?) character. Case III. Their mother, bedridden with a spastic condition of the lower limbs.

Case I.—John Brand, æt. 21 (at date of first observation), single, a carpenter by trade, became an out-patient under Dr. Bastian at Queen-square on Oct. 28, 1884. Dr. Bastian noted the ataxia, the absence of pains, and the comparative youth of the patient, and seeing the interest which I took in the case, very kindly transferred him to my charge.

He complains solely of weakness in the legs. He is a slight, somewhat pale and sallow man; gait unsteady, especially when he turns round; he is unsteady when he stands with his feet together and eyes shut. Hands said to be unaffected, but on closing the eyes, he cannot very readily touch the tip of the nose with his finger. Speech slightly hesitating and occasionally indistinct. Ocular movements normal; no nystagmus; pupils moderate size, act to light and accommodation. Optic discs normal. Legs fairly nourished, strength of muscles fair. Patellar tendon reaction absent on both sides. Cutaneous sensibility of soles and feet normal in all modes; no numbness or paræsthesia. Special senses apparently normal.

History of illness.—Was well till three years ago. Then had some sort of feverish attack, which he describes variously as "bilious fever," or "rheumatic fever." He was confined to bed for seven or nine weeks: was in a great heat and had pains all over him, but the joints were not red or swollen. On recovery he noticed the unsteady gait. He further seems to have had "a peculiar sensation" "which he cannot describe" from the legs up

[1] No. 25.

[2] 'Brain,' No. 35.

to the waist. The unsteadiness has gradually increased, especially during the last twelve months. A year after the illness mentioned above, he had scarlet fever, but this made no difference to his walking. Recently he has had attacks of nausea with retching, but he does not actually vomit, and the nausea passes off in three minutes or so.

Ever since the first illness he has been subject to pain in the limbs, which he thinks is rheumatic; it comes on only when he moves, and does not appear to have the characters of lightning pain.

Never diplopia; never difficulty in micturition (except occasional pain from "gravel"), nor of defecation. No history of syphilis. Ordered Syr. Ferri Iod. ʒj; Pot. Iod. gr. v., ter die.

January 24, 1885.—Visiting him at his home, I was enabled to verify the observations made when I first saw him. There is now, however, no noticeable abnormality of speech, and he says himself this has improved. His hands he says are quite well, and he can do his carpentering work.

Farado-contractility of muscles in lower limbs tested and found normal. Plantar reflexes present. No deformity of feet. The teeth are peculiar—the upper incisors being small, somewhat set apart, the edges worn and somewhat honey-combed. The deformity is very slight, and suggests the so-called mercurial or rickety teeth, not those of congenital syphilis.

On *July* 3, 1885, he became an in-patient at Queen-square under Dr. Ramskill, and remained till the end of August. Dr. Ramskill kindly permits me to utilise the in-patient notes taken by our then resident officer, Dr. James Oliver.

The unsteadiness, which on admission as in-patient was worse than when I saw him in January, continued to increase. Slight unsteadiness of the head, and slight tremor of the legs was noticed. Nystagmus, lateral in direction, was observed, though only from time to time, and only when he looked to the extreme right or left. He complained sometimes of the legs feeling stiff, and of pains in the legs which seemed to draw them up, "cramping pains." There was slight hesitation and thickness of speech. He had some attacks of pain in the abdomen passing into his legs, and lasting a day or two; accompanied with nausea but no vomiting. A purgative given for one of these attacks upset him very much. Once he complained of a girdle-sensation. While in the hospital he was treated first with conium juice, and afterwards with quinine and iron.

July 30, 1886 (at his home).—His general health, which had rather fallen off while in London, has now improved. But the ataxia of the lower limbs has increased; he can now hardly get about the room unless he holds the furniture. He feels as if he had a sponge under his feet, or as if the floor were loose. Romberg's symptom is now well marked; thus when stripping for the examination of his chest, he has to sit down for fear of falling while he is pulling his shirt over his head. Beyond the ataxia, I can find nothing wrong with his lower limbs. The upper limbs

seem normal in every way; he can now touch the tip of his nose with the eyes shut. The speech, however, is rather indistinct, and the nystagmus more decided.

I have not seen him since, but a sister-in-law reports that his legs get no better, though in other respects he is well. He has now been taking nitrate of silver for a long period.

CASE II.—Linda Brand, sister of above patient, single, age 28 (1885).

January 24, 1885.—Seen at the family cottage; she was in bed on account of the severe cold. The history is partly from her own statements, partly from those of her mother, who was then alive.

According to her mother, she had weakness of the legs from the age of 5 years. It was noticed as she came home from school, that her legs became crossed, and that she staggered about. Eleven years ago (age then 17) she had scarlet fever, and this, to use her own expression, took her quite off her legs. She has been worse ever since. But she states that she can still stand and even walk a few steps with support. Ever since the fever too she has been unable to guide her hands, and her speech has seemed to her mother to be odd. With respect to this, she says herself that she has certain "attacks of vomiting which make her speech worse." In these attacks, she vomits phlegm for two or three days: anything that she takes would come up directly, and she therefore abstains from food. At these times also she aches all over, especially in the left arm down to the fingers. The attacks may come on, now, as often as once a fortnight, but less frequently in the summer than in the winter. They have been as frequent as this during the last three or four years, but she had them before that. During the last three or four years also she has had pains "like something biting her legs." During the last two or three years occasional incontinence of urine.

No numbness nor anæsthesia; never diplopia; sight said to be good, except for reading long together (error of refraction?). No deafness, at least to ordinary conversation.

Present condition.—She is of a pale sallow complexion, dark hair and eyes; mouth wide and slightly gaping, teeth prominent; the lower part of the face has a vacant expressionless look. (Her facial aspect strongly reminded me of another young woman with the same disease who was then under my observation.) Speech somewhat indistinct, words brought out with effort and pausing, syllables slurred sometimes. Tongue protruded straight, without tremor; slight tremor of facial muscles as she shows teeth. Well-marked nystagmus, chiefly as she fixes or follows; motions of eyes with this exception normal. Pupils moderate size, act normally to light and accommodation.

Upper limbs very thin, movements of hands and fingers (*e.g.* in trying to unbutton her dress) markedly ataxic; with eyes shut, cannot touch tip of nose with forefinger. No tendon-reaction on tapping radius.

Lower limbs thin; some muscular twitching in region of vasti interni just above knees, but chiefly, she says, "when she thinks

about it." The left leg she can scarcely raise from the bed; but she can raise the right, and tells me she can even bear weight on it when assisted to stand. No rigidity of legs. No deformity of feet. Tendon reactions absent at knees: plantar reflexes not obtained. Sensibility of feet (so far as I could ascertain) normal: muscular sense apparently normal—she knows how her limbs are placed in bed. Reaction of muscles to a moderately strong faradic current normal, but the current is felt very little in the left leg, albeit she feels the warmth and the touch of the electrodes. Spinal column (which could only be roughly examined) appears straight.

About eighteen months later, having heard that she was worse, and had become subject to alarming attacks of choking, I visited her again.

July 24, 1886.—She is now sitting in a chair, and is not in bed, but says she has grown so far worse that she cannot walk, and can only stand a very little. The expressionless look about the lower face is very marked; nystagmus unchanged; pupils as before normal; speech I think more defective, slow and rather drawling, words indistinct and yet somewhat separated; no definite tremor of tongue or lips. Further she says she has become subject to loss of voice: her voice she says "goes off." This is apparent as she speaks now, for sometimes as she begins a sentence, her voice gets squeaky, and subsides into a whisper before the end. There is now marked paralysis of the lower limbs; thus she can hardly move her feet off the stool (paralysis of flexors of hips and extensors of knees); neither can she dorsi-flex the feet or toes at all. I cannot however find any trace of deformity or contracture; she states that the toes "draw under," but this is probably the effect of the extensor paralysis. There is now I think actual wasting of the vasti, in addition to the leanness of the whole lower limbs. Sensation still quite normal. The hands are more ataxic than before; she cannot cut up her food, but can manage to feed herself with a fork. Complains of no pains. Has no trouble with the sphincters. So far as I can make out the spine is still straight, but the right scapula and the posterior part of the right ribs project in a manner that suggests a spinal curvature.

Coming to the "choking attacks," about which I had been told, she says that sometimes, and especially when she lies down in bed, she feels as if she could not breathe, and has to be held up. While talking to me something of the kind came on; she leant back in the chair with her head extended, then over one arm of the chair with her head drooping to that side, appeared to hold her breath, looked distressed and as if about to weep. Her pulse was easily felt at the beginning, but grew feeble towards the end of the attack. She came round in two or three minutes. No actual dyspnœa, no cyanosis, no loss of consciousness, nor any kind of convulsion. Except for the condition of the pulse and of the heart (see below), it might have been a sort of hysterical attack. These attacks she has had for six months, and worse since she was upset by her mother's death (in March of this year). The vomiting

attacks which she described to me on the occasion of my last visit still persist; the last visit was about three weeks ago.

An attempt to examine her larynx was altogether unsuccessful, as she was much too irritable and nervous.

On examination of her heart, I found well-marked evidence of disease, viz. apex beat just beyond nipple-line, in fifth interspace: action rapid: a systolic murmur, mostly musical, heard all over the præcordia, loudest at the left fourth interspace near sternum, and loud along all left edge of sternum, moderate at the apex beat and over aortic area, not audible behind. Probably this murmur was of very long standing, for ten years ago a medical man who examined her heart told her she "must have had rheumatic fever." She never had it to her knowledge. Other possible sources of cardiac mischief suggest themselves, either the scarlet fever, or what would fit in with the nature of her nervous disease—congenital malformation.

The father of these patients, William Brand, was at the time that I saw him (January, 1885) a healthy old man, who, with the exception of chronic rheumatism, had never had any illness in his life. His patellar tendon-reactions were normal. There appeared no reason to suspect him of alcoholism. About his ancestry I have no information. He died rather suddenly on June 8th, 1887, while sitting at table. He had previously, nevertheless, been suffering from dropsy.

More interest attaches to the case of the mother and to her family. I saw her but once; owing to her helpless state and the bitter coldness of the weather, it was impossible to make a thorough physical examination; but I was able to ascertain some facts of importance.

Case III.—Mrs. Brand, age 64 (January, 1885). She gives a clear account of herself and her family. She was never ill till after her third confinement (age then 29 or 30), when she had a white leg. The present affection began to develop from twenty to thirty years ago (and therefore in no immediate relation to the white leg); she speaks of it before it laid her up as "a weakness." Twenty-five years ago she became subject to a feeling of loss of power in the right side; this feeling began in the great toe and passed up that side. It was momentary, but made her drop what she was holding; she has had it on and off ever since. For a period of eighteen years she attended at the Hertford Infirmary; she experienced difficulty in walking; she felt tired and used to fall about. She was also subject to "illnesses" which would take her quite off her feet. In these "illnesses" retching would come on suddenly, and last for some hours; she had abdominal pain at the same time. For some years, in addition to the weakness she felt a numbness right up to the navel; it felt as if she were walking on stumps. Sometimes she did not know when she passed a motion. This numbness has now quite disappeared. During all this period she had no notable pain.

Six years ago, an accident happened to her which seems to have formed a critical point in her disease. She stumbled and fell upon

her back, and has been much worse ever since. She has been unable to stand, and can only lie in bed or sit on a chair: she has also become what she calls "contracted"; the ribs of the left side are, she says, displaced, and she is subject to a dragging pain in the back and side. After the fall she used to have convulsions of pain in the right side, which seemed to "draw the side." From time to time she has difficulty in holding her urine; defecation normal, though the bowels are rather confined.

Present condition.—She is a healthy-looking woman in the face, though thin; at present is in bed. Speech quite natural. Sight and hearing said to be natural. No nystagmus: the state of the pupils unfortunately is not noted, but if I remember right they acted naturally. *Upper limbs.* Right grasp very feeble, left natural. No ataxia of movement; touches nose with finger-tip, even with eyes shut, fairly readily. Tendon-reactions present at both wrists. Right shoulder-joint stiff, cannot raise it above her head. She says this came on after the fall; whether it were due to adhesions, I did not determine. No rigidity of elbow, wrist, or fingers. *Trunk.* A marked projection of the ribs in the left infra-mammary region very suggestive of spinal curvature. Yet, so far as I can make out through her wraps, the vertebral column is straight. She says herself the back is not crooked, but the ribs she was told were displaced by the fall. She can sit up in bed when raised, but cannot get up into that position by herself. *Lower limbs* rigid, partially flexed at hips and knees, and the knees pressed together. She cannot move them from this position herself, and they offer resistance to passive movement. Tendon reactions present at both knees, and well-marked ankle-clonus present on both sides. The muscles both above and below the knees are small; I could obtain no faradic reaction, but had not a battery powerful enough to test this point thoroughly. Sensation roughly tested appeared normal in the feet. She has a dragging feeling in them which distresses her much, especially in cold weather.

This patient died in March, 1886; I suppose from some form of lung disease. She had had a bad cold and cough; and while convalescing from this sat out of doors in her chair. It was thought that she got a chill in this way, for she had to go to bed again, and died in five days' time.

I may say at once that I record the above case as being the mother of two typical cases of Friedreich's disease, herself suffering from some form of spinal disorder. I do not say that her symptoms were those of Friedreich's disease. There was no ataxia, no nystagmus, no speech-affection (although the disease was of long standing); and on the other hand, there was a spastic condition of the lower limbs, with preservation of tendon reactions, and with ankle-clonus. It ought to be mentioned, however, that the woman thoroughly identified her own disease with that of her children; there are indeed some points in her history—e.g. the "illnesses" or vomiting attacks—which correspond with the history of her daughter's case, and it is by no means impossible that the fall on the back to which she attached so much importance may have

set up a myelitis calculated materially to modify her original symptoms.

But this much at any rate may be fairly argued, that the tendency to spinal disease in the Brand family came through this patient. For she was William Brand's second wife; and whereas by his first wife he has three living children all healthy, by this his second wife he has three living children, of whom two are paralysed. And again there is no record of nervous disease on his side of the family; but in her family was a marked tendency to paralysis. She gave me the following account of it.

Her father, William Savill, who died at the age of 70, had during the last twenty years of his life a "weakness" which she thinks resembled her own. He dragged one of his legs. His hands were unaffected. He used to complain much of "rheumatism."

Her mother had three "paralytic fits," in the last of which she died. These, so far as I could make out, were apoplectic: she distinguishes them, at any rate, from her father's illness, "which did not come on in this way at all."

She has two sisters of whom she has lost sight, but she never heard that they were in any way paralysed.

She had several (five ?) brothers. One of these died at middle age in a fit. Another, who died at 67, was for two years before his death "paralysed on the brain." He could not talk well, and his memory used to come and go. But he was never paralysed in his limbs, like herself.

I append a table of all the members of this family concerning whom I have been able to obtain information:—

Date, 1885.

WM. SAVILL (paralysed in legs). m. MRS. SAVILL. (apoplectic).

Male (died in fit). | Male (aphasic ?). | 2 females. | 3 (?) other males.

Linda (*paralytic, Case III.*).

WILLIAM BRAND, married (first) JANE SELL, and by her had

William. | Ann Louisa (Brinlow) (healthy, normal tendon-reactions). | Caroline (Black).

4 children, æt. 21—7 (living and healthy). | 3 children, who died young. | 4 premature births.

WILLIAM BRAND, married (secondly) LINDA SAVILL (*Case III.*), and by her had

Alice (Smith), æt. 32, twice married; her eldest child has left hemiplegia. | Linda (*ataxic, Case II.*). | John (*ataxic, Case I.*). | 7 children, who died young; one of "brain fever"; another had fits.

The first two cases illustrate one special peculiarity of this disease, viz. its tendency to attack several members of one generation. The third case shows that their proclivity to spinal disease was inherited from the mother, having been in all probability inherited by her from her father. It need not perhaps surprise us to find symptoms of lateral sclerosis in the mother, as opposed to the symptoms of posterior sclerosis in the children, if we remember, that such knowledge as we possess of the morbid anatomy of the disease goes to show, that both lateral and posterior columns are affected in course of time, albeit the posterior are affected primarily and most completely. Other instances too are on record of spinal disease attacking several members of a family, yet under various forms.[1]

Nevertheless it is an interesting question, whether this particular form of ataxy cannot, in addition to appearing as a "family" disease, i.e. among brothers and sisters and cousins, be shown also to be directly and strictly inherited. Now in a certain number of instances there has been reason to think that ancestors of the patients were ataxic;[2] but generally this has been a matter of history, not of the author's own observation, so that the exact nature of the ancestral disease remained doubtful. Vizioli,[3] however, has settled the question in the affirmative. A man begat eight ataxic children. One of these cases Vizioli studied, and diagnosed as Friedreich's disease. This same patient of his begat two children, both of whom were under Vizioli's observation, and were the subjects of Friedreich's disease. Here therefore is evidence of direct transmission.

Taking "heredity," however, in the broad sense of family proclivity, there can be no doubt that it is a distinguishing feature of this disease—the feature indeed which chiefly led Friedreich to differentiate it from ordinary tabes. Nevertheless, as cases have been multiplied, it has become evident that the other characteristics of the disease are sufficiently definite to allow of a diagnosis even in sporadic cases; and further that, as might be expected, such sporadic cases do exist.[4] It is important that this should be known, for it is in such cases (especially in their earlier stages, when perhaps difficulty of speech or nystagmus has not yet appeared), that mistakes in diagnosis are most likely to be made.

I have myself seen three instances of sporadic Friedreich's disease; the first under the care of Dr. Gee in St. Bartholomew's Hospital, the second under Dr. Hughlings-Jackson in the London Hospital, the third an out-patient of Dr. Howard Tooth's at the Metropolitan Free Hospital. This latter patient I have twice seen, once with Dr. Tooth two years ago, and again in the present year. Dr. Tooth kindly permits me to publish notes of the case.

[1] "Primary Spastic Paralysis and Pseudo-Hypertrophic Paralysis in Differen Members of a Family." Philip, 'Brain,' No. 32.

[2] See cases by Carré, Rutimeyer, Brousse, Everett Smith, Judson Bury, and the author.

[3] Vizioli, 'La Malattia di Friedreich,' 1885, pp. 11, foll.

[4] See references in Dr. Bury's paper, 'Brain,' No. 34, p. 153; and also 'Archiv. de Neurol.,' March 1887, case by Blocq.

CASE IV.—Bridget Sullivan, age 19 (June 10th, 1885). A healthy-looking girl, though rather small in stature. Her chief complaint is unsteadiness on her legs. She certainly walks very unsteadily, looking at her feet as she goes. She cannot stand well with her feet together, and still less with her eyes shut. Even as she sits, there is a slight unsteadiness of her head. The hands are affected, but much less than the legs: she cannot do needlework as she used to do.

Lower limbs.—Absence of patellar tendon-reactions. Muscles well nourished: the quadriceps cruris contracts well, either when struck or under the faradic current. Sensation appears normal in every way. The left foot turns in a little, and there is here some tendency to club-foot. Otherwise no evidence of contracture.

In the *spinal column*, however, there is a lateral curvature, convexity to the left, in the dorsi-lumbar region; here also is some prominence of the spines (kyphosis?). Her mother too has noticed that she does not hold herself up when sitting. As regards the rest of the trunk, she has occasional pain, of a shooting character, in the left lumbar and iliac regions, but this does not appear to be a prominent symptom.

In the *upper limbs*, Dr. Tooth had noticed a certain amount of ataxia. Thus she could not, with her eyes shut, bring the tips of her forefingers together with certainty, nor touch the tip of her nose quickly.

Eyes.—Movements normal, no nystagmus; pupils act normally; fundus of both eyes natural. Sight is said to be impaired, but apparently she is hypermetropic. There is, I think, a slight peculiarity of *speech*, viz. extra distinctness of syllables and monotony of pitch; but at any rate it is too slight to have attracted the mother's attention. *Mentally* she was, when at school, intelligent and quick; but lately she has become irritable, and sometimes rude to her mother.

Menstruation has only recently commenced.

She has never had any fits, nor vomiting, nor headache, nor indeed any complaint but the present.

History of illness.—It began some two or three years ago. In May, 1883, she was in St. Bartholomew's Hospital, under the care of Dr. Gee. The following is an abstract of her symptoms.

"Ill for a year, with commencing tremors in the hands; drops things when spoken to crossly. Is getting worse.

"Pains in back. When a child, had abscess in the neck. Lateral curvature in dorsal region.

"Cardiac dulness above third rib. Apex-beat in fourth interspace within nipple line. Thrill (?) with apex-beat. Occasional harsh murmur with first sound over second left rib.

"No albuminuria."

A later note (May 2) says: "Tongue tremulous; right arm shakes. Diagnosis, chorea (?)." [1]

[1] It seems to be a fact, that some cases of Friedreich's disease begin with symptoms resembling chorea; *vide* Medico-Chir. Society's 'Transactions,' vol. 68, pp. 149, 152; and Erlenmeyer, 'Centralblatt für Nervenkr.,' 1883, p. 385.

Since that period she has never been what she was before. The staggering gait was first noticed in November, 1884 (about seven months ago).

In June, 1887, I saw her again, after two years' interval. The chief change was a marked increase in the ataxia of the legs. She can now (it is noted) only walk when supported, or by holding on to the furniture. In doing so she sometimes gets her legs crossed, and there is certainly a tendency to throw the feet outwards and forwards. Her mother's expression is that she "throws her feet." She can stand with her feet apart, and can just stand with the feet together, but on shutting her eyes she would fall altogether. Sensation in the feet and legs is still perfect, and she knows their position when in bed. About the feet, especially the left, there is some bluish congestion looking like incipient chilblains. Left external malleolus prominent; left ankle-joint loose, and the foot tends to turn in. Left instep is high, and the toes decidedly tend to turn up. The right foot is well-shaped. I notice that her hands move awkwardly as she fastens up her dress, but she can do this, even fastening the strings behind her back. Sewing or fine work she cannot do, but she can "clean about" as well as ever. Tendon-reactions quite absent, whether at knees, wrists, or elbows. Speech not definitely affected, but it still seems to me a little odd. On fixing an object with her eyes, there is slight shaking of the head; and on fixing to the left, some (doubtful) nystagmus.

She is well-nourished, indeed rather fat; menses regular, breasts well-developed; heart and lung sounds normal.

The family history of this girl stands as follows (1887).

Mother, age 57, an active healthy Irishwoman; never been ill; never touches alcohol in any form, says a glass of beer would make her tipsy. Before marriage, was in good situations as domestic servant. Was one of seven brothers and sisters, none of whom were paralysed. Both her parents were healthy people.

Father (whom I did not see), also Irish, age about 58. Said to be "delicate-looking," but has never been ill. Is a confirmed drunkard. A pledge signed two months ago hangs on the cottage wall, but he drinks still. Comes home on Saturday nights "blind drunk, so that they cannot stop in the house with him." Yet it takes a great deal of liquor to intoxicate him. Sometimes, while in this state, he has pinned his paralysed daughter against the wall, frightening her very much. It should be mentioned, however, that in the two years preceding this girl's birth, he did not drink so much. His family history is unknown to his wife (who was my informant respecting him); but at least she never heard that any of his relatives were paralysed.

These Sullivans have had four children, viz.,

(1) Mary, died in 1867, at age of 2 years and 5 months, of "inflammation of the brain."

(2) Bridget, Case IV., given above.

(3) A son, age 19, healthy. Dr. Tooth examined him and found him free from ataxia, and his patellar tendon-reactions normal.

(4) Catherine, died in March, 1872, aged 1 year and 7 months, of "diseased mesenteric glands." She never walked.

Family predisposition is in this particular instance wanting. But as another possible factor in the ætiology, we ought to note the intemperance of the father. This point has been emphasized by other physicians, and I believe with reason. I have myself had the opportunity of studying Friedreich's disease in four different families, and in three of these, viz. Sullivan, just given, and Harmer and Woodcock, published in the 'Transactions of the Medico-Chirurgical Society,'[1] there has been a history of intemperance in the father. In the first two of these three there was no history of nervous disease among the ancestry; in the third (Woodcock) there was a strong nervous heredity on the mother's side; her father was probably ataxic, her sister insane. But in addition, her husband (as she has since informed me) was a hard drinker during their early married life, and her own notion (perhaps not an irrational one) is that her children's paralysis is due to the frights she sustained during pregnancy from her husband's drunken fits.

I find that parental or ancestral intemperance is mentioned in two instances by Friedreich, and also by Everett Smith, Vizioli, Quincke, Judson Bury, and Kingston Fowler. Some of the histories read almost as if intemperance had started a disease in the drunkard's offspring, which subsequently became hereditary. Thus the grandfather of Dr. Bury's patients had small feet, and could not walk in the dark, and his father again drank heavily; and in Vizioli's observations quoted above, we find that an intemperate man had eight ataxic children, and through one of these two ataxic grandchildren.

I admit, nevertheless, the force of the objections, firstly, that drunkenness may be itself the expression of nervous disease, and secondly, that the parental drunkenness may be a mere coincidence, for intemperance is very common, while Friedreich's disease is rare. But it is likely enough that several factors may be required for its production, and they may differ also in different cases. We know too little of the ætiology to neglect the study of any possible cause.

I have elsewhere remarked, that acute specific diseases appear sometimes to assist in the development of Friedreich's disease, or to cause an aggravation of it. The cases of John and Linda Brand (I. and II.) are curious in this respect. They both had scarlet fever about the same period of their lives, and after the first development of their nervous symptoms. John was none the worse for it; but Linda's disease was so much aggravated that she became a paralytic practically from that date. Nevertheless, John's disease developed originally after some febrile attack of uncertain nature. I should like to mention in this connection the case of two brothers recently shown to me by Dr. Hughlings-Jackson at the London Hospital. They both had Friedreich's disease; in the first the

[1] Vol. 68.

disease had developed in such close connection with diphtheria, that it was for a time questionable whether his symptoms were not due to some diphtheritic paralysis, of a permanent and progressive nature. Then the second brother came under observation, who had never had diphtheria, but exhibited nervous symptoms similar to those of his elder brother.

There is a condition not infrequently found in the subjects of Friedreich's disease which deserves notice—I mean the deformity of the feet. I do not think much attention was given to it before the observations of Rutimeyer, who noted the development of club-foot and a tendency to dorsiflexion of the toes. Dr. Bury describes and gives an illustration of the deformity. In the two Brands (Cases I. and II.) there was nothing of the kind; in the case of Bridget Sullivan (Case IV.) the left foot apparently is becoming deformed. I have watched the condition develop in another family (Woodcock). The instep becomes prominent, and the metatarsals appear to be shortened; at the metatarso-phalangeal joints the toes become over-extended, and at the first interphalangeal joints flexed. The foot looks humpy and shortened. Below, the plantar arch (at least, as seen from the inner side) is abnormally high, and the balls of the toes are very prominent. The foot also tends to assume the position of equino-varus. Perhaps a contracture of the long extensor of the toes associated with a paralysis of the interossei might produce the main features of the deformity. It certainly is not always present in Friedreich's disease, even in advanced cases (Case II.); and I have seen it in other diseases—once in a case of old-standing multiple neuritis, once in two sisters, who, though they could hardly be said to be suffering from Friedreich's disease, presented some symptoms which are worth mentioning in that connection. They were as follows—

A girl, æt. 19, was admitted by me to Queen-square Hospital for difficulty in walking. This, she told me, was due to a deformity of her feet. On examination I found the humpy shortened foot which I have endeavoured to describe above. *The patellar tendon reactions were entirely absent.* The difficulty in walking and the deformity of the feet had come on gradually about the age of 10, and she thought were getting worse. I could discover no other nervous symptoms. A sister, age 30, brought her, and this sister had a similar deformity, but in the right foot only. *The patellar tendon-reaction was quite absent on the right side, and only just present on the left.* The foot, she said, did not impede her walking unless she tried to go fast. On enquiring into their family history, they told me that a brother, aged 23, living in China, had the same shaped feet. There were six others in the family, reported healthy. Further, a maternal uncle had the same deformity of feet, but had no difficulty in walking.

I believe that cases of club-foot occurring in several members of the same family have been reported by orthopædic surgeons; perhaps an investigation of the nervous system might show, that in such instances there existed more than a mere local deformity, as I can scarcely doubt was the case in these patients.

FURTHER LIST OF PAPERS UPON FRIEDREICH'S DISEASE, OR BEARING UPON IT.

CHARCOT: 'Gazette des Hôpitaux,' April 26, 1887, and 'Progrès Médical,' June 1887.
MORTON PRINCE: 'Boston Medical and Surgical Journal,' cxiii., p. 371.
SINKLER: 'Medical News,' July 4, 1885.
ERLICKI and RYBALKIN: 'Wjestnik Psychiatrii,' &c., 1885, No. 1. (Abstract in 'Neurolog. Centralblatt,' 1885, p. 375.)
SAME AUTHORS: 'Archiv für Psychiatrie,' &c., 1886, p. 692.
GOWERS: 'Diseases of Spinal Cord,' pp. 349 foll. (contains description and illustrations of sections of a cord).
BLOCQ: 'Archives de Neurolgie,' March 1887.
DESCROIZILLES: 'Progrès Médical,' 1887, No. 28.
FERRIER: 'Brit. Med. Journal,' June 4, 1887.
FREYER: 'Berlin. Klinisch. Wochenschrift,' 1887, No. 6.
STINTZING: 'Münchn. Med. Wochenschrift,' 1887, No. 21.
DANA: 'New York Medic. Record,' July 2, 1887.
BRISTOWE: 'System of Medicine' (6th ed.) p. 1059.
GOODHART: 'Diseases of Children'

MALARIAL PARAPLEGIA.

BY C. W. SUCKLING, M.D. (LOND.), M.R.C.P

Physician to the Queen's Hospital, Birmingham, &c.

PARAPLEGIA is a very rare and interesting manifestation of ague, occasionally taking the place of the ordinary fit, and occurring regularly in an intermittent manner. It may, however, occur at irregular intervals in those who have been attacked by ague. Romberg first described this condition,[1] and his case was a very typical one, the attacks of paraplegia occurring suddenly on alternate mornings at the same hour and passing off in a few hours. Sensation was unaffected, but the sphincters were paralysed. The attacks were soon checked by the administration of quinine. Harting and Erb have also described similar cases.

Dr. Gibney[2] has described three cases of intermittent spinal paralysis of malarial origin cured by quinine; the attacks were irregular in the time of their onset, the tendon reflexes were lost, and a remarkable feature was the diminution and absence of the electrical response of the nerves and muscles. His cases were also quickly cured by quinine.

Westphal has recorded a remarkable case of periodic paralysis in the 'Berliner Klinische Wochenschrift,' Nos. 31 and 32, 1885, in which all four extremities were affected. The paralysis came on at night and disappeared before morning. The loss of power was absolute, and the nerves and muscles completely lost their electrical excitability during the periods of paralysis. The attacks recurred for several months and finally ceased. There was apparently no malarial taint, and Westphal can offer no explanation of the affection.

A case of intermittent paraplegia, independent of malaria, has also been described by Dr. Shakhnovitch, related in the 'London Medical Record,' 1884.

No mention of malarial paraplegia is made in any of the ordinary text-books of medicine and in several important works on neurology. I find that a brief account of the affection is given in Dr. Ross's treatise, in Bramwell's work on

[1] 'Syd. Soc.,' vol. ii., 1853.
[2] 'American Journal of Neurology and Psychiatry,' vol. i., No. 1, 1882

the Spinal Cord, and a fuller description is written by Dr. Bastian in Quain's 'Dictionary of Medicine.' But no reference is made to the affection in the works of Gowers, Hammond, Hamilton, Wilkes, and Buzzard. It will be readily inferred from the above statements that the affection is an exceedingly rare one, and that little is known of it.

In searching for information concerning the disease I obtained much assistance by applying to Dr. Neale's 'Medical Digest,' in which valuable work references to several recorded cases are given.

The nervous system is largely implicated in ague—so much so that Trousseau considered that the disease was a neurosis.

J. P., a shoe-maker, was admitted into the Queen's Hospital on July 4th last, complaining of loss of power in his legs. There was no family history of any disease. Nineteen years ago the patient had an attack of venereal disease, a sore throat following; and two years later he had ulcers on both legs.

Eleven years ago, while serving as a soldier in India, he had his first attacks of ague; and for three months had an attack of more or less severity every day, until he was moved with the troops up the hills.

Whilst at home, eight years ago, he had two attacks of ague, and a year later he went out to Afghanistan, and for two years suffered from the same complaint (quotidian). He returned home in 1882, and three months after his return he had another attack.

In June, 1886, he had an attack of ague, and a fortnight subsequently he lost his speech for a week. This loss of speech came on suddenly with giddiness, and he was treated at St. Bartholomew's Hospital. The giddiness only lasted a few hours and his speech returned suddenly, but was thick for a day or two after.

At the beginning of August, 1886, the patient had another attack of ague, and about a fortnight after was admitted into the Queen's Hospital, Birmingham, suffering from paralysis in both his legs. When this attack came on he felt as if he had received a blow of a fist on the back of his neck; he felt giddy, lost power in his legs, and fell. He says that sensation was lost in his legs. He remained in the hospital for a fortnight, and power gradually returned. On the third morning he was able to raise his left leg, and on the fourth, moved his right.

Since his discharge from the hospital last year he has had several attacks of ague, each attack lasting a few days; the seizures occurring daily, and being marked by a cold and a sweating stage.

A fortnight ago (June 20th, 1887) he again had an attack,

and the next day had a pain round the middle of his body—"girdle pain"—and on July 4th, with a sudden attack of giddiness, the patient for the second time lost power in his legs, was confused and almost unconscious for about five minutes, and passed his urine involuntarily.

He lost his speech for an hour and suffered from headache. On admission, he was found to have lost all power and feeling in the legs, and his speech was thick and slurred.

On the front of both legs were several cicatrices of a coppery colour, where he had had ulcers some years ago.

On the morning after admission (July 5th) sensation in the legs was found to be perfect, though all forms of sensation were quite lost when admitted. He stated that feeling returned in his legs during the night with a sensation of "pins and needles." Motor power was still completely lost in both legs. The plantar and cremasteric reflexes were absent; the knee-jerk was normal. There was no pain or tenderness in the spine or lower extremities. The muscles of the trunk, head, and neck, and upper extremities, were unaffected. There was no affection of the special senses or of the ocular movements, and no change in either fundus oculi.

The memory was good, and there was no impairment of intelligence.

Speech was a little impeded, there being a slight stammer.

Involuntary micturition was still present, the urine dribbling away; but later in the day he recovered power and was able to retain it.

The faradic response of the nerves and muscles of the lower extremities was normal.

On the morning of July 6th the patient was able to raise his left leg off the bed, and about an hour after was able to do the same with the right leg.

The plantar reflex was still absent; the cremasteric reflex had returned, and the knee-jerk was very lively.

At night the patient was able to walk by himself to the lavatory without help, and could pass his urine naturally.

July 7th.—The patient expressed himself as being quite well, and he wished to leave the hospital.

The feet were cold, but the surface temperature was 97° F. The plantar reflex was still absent.

July 8th.—The plantar reflex was now present on both sides, the knee-jerk was slightly exaggerated, but there was no ankle-clonus.

The patient walked about perfectly well; motion and sensation were perfect. He was discharged perfectly well.

During his stay in the hospital his temperature was normal, as also was his urine.

There can be no doubt, in the above case, that the paraplegia with which he has twice been seized is connected with the attacks of ague from which he suffers. The paralysis each time occurred a fortnight after an attack of ague, was sudden in its onset and more gradual in its disappearance—the left leg recovering power before the right.

Recovery in each attack commenced on the third morning after the onset, and was complete in a few hours.

In each attack, moreover, there was temporary loss of speech, together with dribbling of urine.

The presence of paralysis of the sphincter vesicæ muscle in itself is sufficient to negative the supposition of the paralysis being hysterical in its nature, and the presence of temporary loss of speech and aphasia showed that the lesion was not one limited to the spinal cord.

The patient on his admission was given quinine; but it is impossible to attribute the recovery to the use of this drug—paralysis in connection with ague being intermittent and recovering of itself.

The attack was so short-lived that it was impossible to make a more complete examination than that above recorded, but the patient has promised to attend the hospital at once should he have another seizure.

CONCUSSION OF THE SPINE FROM A FALL FOLLOWED BY WASTING AND PARALYSIS OF NUMEROUS MUSCLES IN BOTH ARMS, GLOSSY SKIN AND LOSS OF SENSATION OF RIGHT HAND.

BY FREDERICK W. MOTT, M.D. (LOND.), M.R.C.P.

(Medical Registrar, Charing Cross Hospital.)

I AM indebted to Dr. Green for kindly allowing me to show this case at the Neurological Society, and to publish the following notes.

George Hunt, æt. 49, a joiner by occupation, sought admission to Charing Cross Hospital, for loss of power in the arms and hands. There was nothing noteworthy in his family history, except that it was free from hereditary taint.

Patient had for many years been a total abstainer; he had never had venereal disease. His work was carriage building; although paint was always about the shop, yet he was quite sure that he had never exposed himself to lead poisoning, for he had always kept his hands very clean, otherwise he would have been fined. Moreover, he had never had colic. Patient stated that up till January 1st, 1887, he had enjoyed perfect health (he was then a heavy man weighing 14 stone), and while out for a walk he slipped and fell backwards, striking the back of his head and neck. He was carried home unconscious, and remained so till the following morning. He kept his bed for about a week, and at the end of this time he got up, apparently not much worse for the accident, for he was able to move both arms and legs, and he suffered no pain. Three days later, or ten days after the fall, he complained of a tingling pain in the right shoulder, afterwards extending to the back of the hand and little finger. The pain continued for about eight hours, and was followed by loss of movement and a feeling of coldness in the hand. He was unable to move his arm or hand. At the end of April, or rather more than three months later, he complained of tingling pain in the central fingers of the left hand, and also in the left shoulder; this was followed by loss of power to raise the

left arm or supinate the left hand; and while the right side has improved a little, this has steadily got worse.

He has not had any visceral disturbances nor any difficulty in walking. He was for some time in the Lincoln Hospital without deriving much benefit.

Present condition, July 14th.—There is loss of sensation on the dorsum of the index finger, thumb and radial side of the middle finger and hand, also of the lower two inches of the radial border of the forearm. The fingers and thumb of right hand are pointed, the nails long and thin, and the skin *smooth and glossy*, especially the dorsal surfaces.

There is extensive wasting of a number of muscles of the right shoulder, arm, and hand. The shoulder is adducted and only very limited movements of flexion, extension, and abduction, can be performed. The elbow is semiflexed and the wrist joint midway between pronation and supination, with ulnar deviation of the hand. The fingers are extended and the thumb slightly flexed. Slight movements of abduction and adduction of fingers can be performed, and the ring and little finger can be slightly flexed and extended at the metacarpo-phalangeal joint.

The muscles of the right side most obviously wasted are the deltoid, triceps, supinator longus, the muscles that flex and extend the wrist and fingers and pronate and supinate the hand. There is a great deal of rigidity about the shoulder, and the arm cannot be raised as high as the head by force. *The left side* is affected in a similar manner to the right with the following *notable exceptions.* There is no loss of sensation and no trophic affection of the skin. There is not quite such extensive atrophy of muscles, as he can supinate the hand and can slightly flex the wrist, also he is able to flex the middle, ring, and little fingers. There is no alteration of the knee-jerks, nor any symptom pointing to a transverse lesion of the cord. The pupils are equal, and respond to light and accommodation.

There is not now, nor has there ever been, any pain or stiffness in the neck; and the spine may be percussed without causing any painful sensation. Warm sponges were also applied without any result.

Electrical Examination.

Faradism.—Trapezius-sterno-mastoid, rhomboids and levators ang. scap. of both sides react normally.

Left arm.—The triceps, deltoid, supinators and extensors of the wrist and fingers and the thumb do not react at all.

Right arm.—The deltoid, triceps, lower fibres of pectoralis maj. do not react, nor do any of the muscles of the forearm and hand, except the flexor-carpi ulnaris, opponens pollicis and first dorsal interosseous.

Galvanism.—The muscles of neck and back react normally.

Left arm.—Deltoid does not react, biceps normal, triceps requires a stronger current than normal. The supinator longus and the long and short extensors of the wrist do not react to the strongest current that can be borne. In the extensors of the fingers AC.C. > KC.C. Ulnar side of the flexor prof. digit. reacts normally.

Right arm.—The deltoid does not react to the strongest current that can be borne. Lower fibres of pectoralis maj. and serratus require stronger current than normal KC.C. before AC.C. Biceps one and a half M.A. In the forearm and hand only those muscles supplied by the ulnar nerve respond.

The patient was ordered the following treatment by Dr. Green. To be galvanised every day, to have massage of the affected limbs twice a day. A mixture was given of which 5 grs. of iodide of potassium was the principal ingredient.

The patient slowly improved on this treatment, and in October he was able to raise his right hand above his head and place it on the back of his neck. The rigidity and stiffness in the elbow and joints had disappeared; the movements of the right hand and arm were still very limited, but considerably improved. The loss of sensation and the glossy condition of the skin of the right hand still existed; there was less improvement of the left hand; if anything, flexion of the fingers was less extensive than when admitted.

The following are the results of various electrical examinations made by Dr. Murray and his assistant:—

Oct. 20th, Faradism.—*Left side.*—The deltoid, extensors of wrist and fingers, and abductor pollicis still fail to react to any current that can be borne.

Of the other muscles the supinator longus requires the strongest currents, and all the others act sluggishly.

Right side.—The reactions in the main similar to those on the left, except that the deltoid now reacts, and the small muscles of fingers and thumb are less excitable than on the left side.

Oct. 28th, Galvanism:—

Left side: Abductor indicis react to 1½ MA ACC = KCC. At 3 MA ACC > KCC.

2nd dorsal interosseous, KCC obtained at 1½ MA. At 3 MA KCC > ACC.

3rd dorsal interosseous, KCC at 2 MA At 3 MA KCC > ACC.

4th dorsal interos., KCC at 1½ MA. At 3 MA KCC > ACC.
Supinator longus, weak sluggish contraction at 2½ MA. KCC > ACC.

Nov. 3rd :—

Right arm: Abductor indicis, KCC at 1¾ MA; 1st ACC at 2¼ MA.
2nd dorsal interos., KCC = ACC at 1½ MA.
3rd ditto KCC = ACC at 1¾ MA.
4th ditto KCC = ACC at 1¾ MA.
Opponens pollicis, ACC and KCC at less than ½ MA; contraction sluggish, ACC > KCC.
Adductor pollicis, KCC > ACC, KCC. 1st at 3 MA.

Nov. 20th :—

R. deltoid, 1st at 2½ MA; at 4 MA ACC > KCC.
L. deltoid, 1st contraction (KCC) at 6 MA.
Biceps r. and l., 1st KCC at 1 MA. KCC > ACC.
Pectoralis maj., r. and l., 1st KCC at 3 MA; KCC > ACC.

Extensors of Forearm :—

R. KCC at 4½; ACC > KCC.
L. ACC at 3½; ACC > KCC.
Supinator longus. L. ACC at 4½; ACC > KCC.
ditto. R. ACC at 5½.
Flexor sub. digit., R. ACC at 3½: ACC > KCC.
ditto. L. ditto,

Dec. 28th.—Patient can now raise both arms to the top of the head. The small muscles of the hand do not seem to have recovered to the same degree as the larger muscles of the limbs. The anæsthesia and trophic condition remains unaltered.

Comments.—The diagnosis as to the pathological lesion is obscure. When the case was shown at the Neurological Society, various opinions were expressed. Dr. Green and myself were of opinion that it was a concussion myelitis, in which the grey matter was the seat of subacute inflammation, the anterior horns of the cervical enlargement being more particularly involved. The loss of sensation and trophic disturbance we thought possible to account for by the posterior horn being slightly involved. We adopted this theory rather than meningitis, affecting the roots of the nerves, on account of the complete *absence of rigidity of the neck and of pain*. Dr. Wilks was inclined to agree with this supposition. It was urged by Dr. Ferrier that it was unlikely for the grey matter alone to be affected, and he did not think this could occur without giving rise to affection of the conducting paths of the cord, and therefore paraplegic symptoms; he was, moreover, of opinion, that the irregular distribution, the loss of sensation,

and the trophic condition of the skin, rather pointed to an affection of the nerve roots of the nature of a meningitis.

Dr. Hughlings-Jackson expressed the same opinion, although he admitted that it was difficult to understand why the patient had no pain or stiffness of the neck.

Dr. Gowers, in his work on 'Diseases of the Nervous System,' says when the damage involves the grey matter, the extent of the muscular wasting varies greatly; it seldom affects both arms and legs, but it is usually irregular in distribution—sometimes widespread, sometimes limited; and he cites several cases, none of which correspond to the one described here.

This case is interesting, whatever be its pathological condition, as showing that a fall on the back may be followed in a previously healthy man by muscular wasting, and other indications of severe affection of either the spinal cord or nerve roots without any evidence of disease of the vertebræ.

The slow but satisfactory progress of the case is also important, as it shows that a hopeful prognosis may sometimes be made after spinal concussion which the condition of the muscles at first sight would hardly warrant. I do not, however, expect that he will recover the power of executing the finer movements of the hand, as so very little progress has been made so far.

NOTES OF A CASE OF ATHETOSIS ASSOCIATED WITH INSANITY.

BY T. DUNCAN GREENLEES, M.B. (EDIN.)

City of London Asylum, Stone, Dartford, Kent.

THE clinical study of obscure nerve disease is always one of interest, and not infrequently results in the elucidation of facts regarding the functions of the nervous system previously more or less unknown.

Athetosis is one of these conditions concerning which our knowledge is at present limited, and observers are still at variance regarding the situation of the lesion which gives rise to such peculiar symptoms. It is to Dr. Hammond, of New York, that we are indebted for the first scientific description of this disease; antecedent to this, however, the symptoms were not unknown to several French neurologists, although they failed to appreciate the importance of their knowledge.

During the past ten or fifteen years, cases have been described in this country by Gowers, etc.; and Oulmont has written a monograph on the subject. I venture to submit a few notes I have made, of an interesting example of this affection at present under my care.

JOSEPH L. first came under notice in this Asylum in 1883, being at that time in his thirty-second year; when he was admitted he laboured under various insane delusions, and exhibited the mental symptoms of secondary dementia: his memory was enfeebled, and his expression noted as vacant. He was of strumous diathesis, was somewhat paralysed on the left side, and the muscles of his arms and neck were affected by peculiar rhythmical contractions.

From the various notes regarding him in the Case Books it would appear that, since his admission, he has been quiet and well conducted as a rule, rarely excited, but at all times somewhat emotional and even lachrymose, more especially when the question of his discharge or removal is discussed with him, as he expresses no desire or anxiety to leave the Asylum. The paresis and muscular contractions described as existing at the time of his admission have never been so severe as to interfere with his usefulness in the wards, or even taking a fair amount of exercise beyond the Asylum estate.

Family History.—The patient states that both his parents are dead, his mother having died of "tumours"; he does not recollect his father, who died when he was a child. He has two sisters and one brother alive and reported to be in good health. There is no hereditary predisposition to any of the neuroses known in his family.

History of Present Illness.—Previous to the year 1870, the patient had always enjoyed excellent health, but during the winter of that year he got wet while following his avocation of labourer, and contracted, what he thought to be at the time, a severe cold. He immediately went to bed, and next day, on attempting to rise, found that he had lost all power over his limbs, and was surprised to observe that he was unable to speak. For some weeks afterwards he was confined to bed, and by degrees the paralysis passed off, but, as he regained power in his limbs, he first noticed various peculiar movements of his muscles over which he was unable to exercise any control whatever. These movements commenced in the legs and feet, then ascended to the arms and hands, and finally attacked the muscles of his head and neck. Three or four months after the onset of the paralysis his speech returned, but has ever since been imperfect. He attended in succession several London hospitals, where he was treated with nourishing diet and tonics with some benefit, he thinks, to the symptoms. He has never undergone a course of electric treatment. Previous to his admission to this Asylum he was under treatment at Caterham Asylum, but he is unable to give a satisfactory account of the attack of insanity from which he then suffered, as he says he remembers nothing of why he was sent there or of his mental condition at the time.

Present Condition.—The patient is a man of medium height, of fair complexion, and with sandy-coloured hair and beard. He has had disease—probably strumous—of the left elbow, which has resulted in ankylosis with the joint in a semi-flexed position. He has a peculiar shuffling gait, trailing his feet slightly; when walking, the head is thrown back and his body sways from side to side; this is apparently due partly to slight lateral dorsal spinal curvature and consequent weakness of the spinal muscles. When standing, his customary attitude is with his head well thrown back, his right arm hanging by his side, and the left hand closed, flexed at the wrist-joint and pressed firmly against his side close to the groin. Movements of the left forearm are limited in extent, and pronation and supination are nearly impossible, owing to the existence of the elbow-joint disease.

While conversing with him and watching him carefully, the fingers of the left hand are observed to separate slowly; then one by one, commencing with the littte finger, they close upon the palm, giving to the hand a characteristic claw-like appearance. Generally the thumb is drawn towards the middle line, and is then inserted between the first and middle fingers. These movements take place by flexion at the metacarpal, and latterly at the phalangeal joints, and are slowly and rhythmically performed.

At the same time the hand is gradually turned round, so that when the movements are completed its palmar surface is nearly uppermost. During sleep the movements cease, and, although always present, they are much more marked when his attention is directed towards them. Similar slowly performed muscular contractions affect the right hand, but they are not nearly so defined as those affecting the left hand.

The instep of the right foot is unusually prominent, and when the patient's attention is withdrawn, or when he is exerting himself in any way, the foot is observed to become slightly flexed at the ankle joint, and at the same time bent inwards so as to form a temporary talipes varus. The great toe is at first extended, forming nearly a right angle with the other toes; it is then drawn towards the mesial line, and partially overlaps the other toes, which are being slowly flexed on the sole of the foot. These movements of the right foot and toes occur almost simultaneously with those affecting the left hand. The left foot performs similar muscular contortions, but, like the right hand, they are much more limited in extent and less defined than those affecting the corresponding limb.

While conversing with the patient, it becomes at once evident that his speech is affected—there is a hesitancy observed; while speaking, his head is suddenly jerked back, and the facial muscles undergo various contortions in his efforts to articulate. At the same time the superciliary muscles contract, giving his eyeballs an unusual prominence: and he stutters indistinctly and incoherently several times before he succeeds in his efforts to speak. The words are ill-pronounced; he occasionally slips a letter or a syllable, and until accustomed to his peculiar mode of articulation it is difficult to understand what he says. This condition more closely resembles that found in persons with what is popularly called an impediment in their speech, than the slip-shod articulation so characteristic of persons suffering from general paralysis of the insane.

His muscular system is fairly well developed with the exception of the muscles of the left arm, which are markedly and generally wasted. The following table shows the extent of this atrophy, which is the more evident when compared with the muscles of the right arm.

Circumference of both Arms at Different Levels.

Right Upper Arm.	inches.	*Left Upper Arm.*	inches.
Upper third . . .	11	Upper third . . .	8¾
Middle third . . .	10½	Middle third . . .	7¾
Lower third . . .	9	Lower third . . .	7
Right Fore Arm.		*Left Fore Arm.*	
Upper third . . .	10¾	Upper third . . .	8¾
Middle third . . .	10	Middle third . . .	8½
Lower third . . .	6½	Lower third . . .	5¾

Although the left arm is so much atrophied, muscular power appears fairly good, and the grip of both hands is about equally strong. How far this atrophy is due to the joint disease and consequent disuse of the limb, and if there is any connection between the nerve disease and it, it is extremely difficult to say; in this place it may be well to mention, that hypertrophy of the affected muscles has been frequently found in well-marked cases of athetosis. The various muscles respond well to electric stimuli, and contractility appears normal on both sides.[1] The patellar tendon reflexes are distinctly exaggerated on both sides, more especially the left, but I was unable to obtain ankle clonus on either side. The various cutaneous reflexes are unusually brisk, and tickling the soles of his feet causes the patient to wriggle about and struggle very much. Ophthalmoscopic examination of the eyes reveals nothing abnormal, and there is no alteration of temperature in either extremity. The other senses and the various systems present nothing worthy of note. There is no evidence of venereal disease, and the patient states he has never had syphilis.

REMARKS.—This case resembles, in many respects, one described by Dr. Sharkey.[2] In his case, however, the symptoms followed infantile convulsions succeeded by hemiplegia, and the mental faculties were always good; whereas in my case it is evident that the muscular contortions followed upon an attack of hemiplegia when he was twenty years of age, and mental impairment did not show itself until some time afterwards. In fact, so far as I am aware, this is the first case of insanity recorded succeeding on well-defined athetosal symptoms. Some years ago Dr. Fletcher Beach[3] described several cases under his care in imbeciles, but recently he has had an opportunity of examining the brain of one of his patients and there discovered a cortical lesion. He, however, has been induced to alter his opinion regarding the diagnosis of this case, and thinks it was an example of what is termed "post-hemiplegic choreiform movements."

Dr. Gowers[4] states that the symptoms in many such cases point to a partial recovery of those nerve cells, whose functions have not altogether been destroyed, after a primary hemiplegia. Dr. Bastian[5] is of opinion, that the condition termed "athetosis" is merely a variety of post-hemiplegic chorea, and seems to accept the theory of Oulmont, who maintains that the symptoms are due to what he calls "athetosic fibres" which

[1] This points to the atrophy being the result of the ankylosed joint, and not to any changes in the muscular fibres the result of nerve disease.

[2] 'Brain,' April 1885, p. 85.

[3] 'British Medical Journal,' 1880; 'Journal of Mental Science,' July 1887

[4] 'Diagnosis of Diseases of the Brain,' p. 61.

[5] 'Paralyses: Cerebral, Bulbar, and Spinal,' 1886, p. 192.

are supposed to exist in the posterior part of the internal capsule, a portion of the brain supplied by a special branch of the sylvian artery, the rest of the capsule deriving its main nourishment from a branch of the posterior cerebral.

We are still, however, very much in the dark regarding the localisation of the disease, but it appears to me that the weight of evidence, derived from clinical and pathological research, points to a lesion more deeply situated than the cortex of the brain. Injury to the motor fibres of the internal capsule explains most of the symptoms in my case, and if the sensory fibres remained intact this would account for an entire absence of hemianæsthesia which, according to Ferrier,[1] always exists after gross injuries or even pathological conditions of this portion of the brain.

[1] 'Functions of the Brain,' 2nd edit. p. 324.

NOTES ON A CASE OF AMNESIA.

BY J. W. BATTERHAM, M.B. (LOND.), F.R.C.S.

THE subject of these remarks is a lady aged 60. Three years ago, after the death of her son, she had an attack of right hemiplegia, from which she recovered in a few weeks. Since that attack she has suffered from diabetes, and sugar is still present in her urine, whenever she abandons her usual (moderately restricted) dietary.

In January of the present year (1887) a recurrence of the hemiplegia was feared, as the patient suffered for three or four days from insomnia, violent pain in the head, giddiness and marked confusion of her mental faculties. After free purgation these symptoms passed away, leaving the patient in her present amnesic condition.

She now (April 1887) feels in good health, is cheerful, and takes walking and carriage exercise. Her appetite is good, tongue clean but rather dry, bowels costive. The urine has a sp. gr. of 1020 and is free from albumen and sugar.

The functions of the cranial nerves are normal. There is no apparent loss of sensation or any paralysis of the limbs, except, perhaps, a slight tendency "to bear to the right" when patient is walking out of doors.

The patient can hear perfectly. Her friends think that her hearing is even more acute than formerly. She understands everything that is said to her, and can converse readily, though often "at a loss" for a name (proper or common), for which she either unconsciously substitutes another, or intentionally uses a periphrasis.

She can repeat from memory words learned long ago, and can correctly describe long past events, but her memory for occurrences of recent date is greatly impaired, so that she often cannot recollect what she did a few days ago, or even what she ate at her last meal.

She can repeat readily whatever is said to her. Her sight is good. She can, with glasses, distinguish and count small dots on a sheet of paper when they are placed about a centimetre apart. She cannot, however, read written or printed characters, and is unable to name the letters of the alphabet when they are

pointed out to her without making numerous mistakes, but if asked to point out letters named to her, her blunders are much less frequent.

Numerals are recognised singly and in combination. The date of the year was at once pronounced by her, on seeing it written. She recognises common objects, but has more difficulty in naming them when they are presented to her for that purpose than she has in recalling the names she uses in ordinary conversation. On being shown a *doll*, and asked to name it, she called it "*a book*," and when told that she was wrong, guessed "*album*," "*dictionary*," &c. Asked if it were a baby, she shook her head, but readily assented when the word "*doll*" was suggested to her, saying, "of course, it is a doll." In no case was the patient able to *write* the name of an object, though unable to *utter* it.

Recognition of objects is facilitated by allowing patient to handle, smell, or taste them.

She writes well, either from dictation or spontaneously. A letter of four pages, written to an absent daughter, was perfectly intelligible, except that the word "*chain*" had been substituted for some other substantive of dissimilar meaning. Patient had also addressed the daughter and grand-daughter, to whom she was writing, by the names of the daughter and grand-daughter (respectively) with whom she was living. *She cannot, however, read aloud a word of what she has just written, nor is the writing intelligible to herself.* By the time she has arrived at the end of her letter she has forgotten the beginning. If interrupted in her writing, what she has already written must be read to her before she can resume the thread of her narrative.

She copies print or writing, but without knowing the meaning of what she writes. She has great difficulty in transliteration from printed to written characters, or vice versâ.

Occasionally, after copying a short word she will spell it over aloud and pronounce correctly, understanding its meaning; but cannot always manage this. When asked to copy the words, "OUR," "SISTERS," "KATE," she did so *correctly* in legible printed characters, but more as if she were *drawing*, i.e. she did not form the different portions of the individual letters in the same order as a person generally does, and began one letter before she had finished the preceding, returning to the unfinished one afterwards to put in the "finishing touches." On being asked to spell what she had copied, she pronounced the first word O, U, *T*, and the last *Z*, A, T, E. She also miscalled the S in "sisters." In short she miscalled three of the four consonants before her, but repeated the five vowels correctly. Tested with different words a few days afterwards, she still remembered

the vowels, but made several mistakes with the consonants. Though the act of copying *printed* characters seemed to render the patient but little assistance in her recognition of them, the contrary held good with regard to *written* symbols. When asked to spell out a word written in the "round-hand" of the copy-books, she failed with several letters; but on being told to copy the unrecognised signs, or to run her pencil over them *as if writing them*, she in most cases recognised their name and significance. This experiment was repeated several times, and the patient was delighted to find that she could "jog her memory" of letters in this way.

The recognition of those printed letters, *the form of which was closely allied to that of the corresponding written symbol usually employed by her*, was also facilitated by this device.

The condition of this patient is of some interest, when considered with reference to the current views of the cerebral mechanism by means of which written and spoken language is appreciated, and by means of which we are enabled to express our thoughts in spoken and written words.

The theory by which the foregoing case seems best explained is that held by Dr. Charlton Bastian,[1] which agrees in many points with that enunciated by Prof. Kussmaul in 'Ziemssen's Cyclopædia,' vol. iv.

It supposes the presence in the cerebral cortex of—

(1.) A centre for the perception of the *sounds* of spoken words—the auditory word-centre.

(2.) A centre for the perception of the forms of written or printed words—the visual word-centre.

These two "word-centres" are probably parts of the perceptive centres for Hearing and Sight, localised by Ferrier in the upper half of the superior temporo-sphenoidal convolution, and in the angular gyrus and adjacent part of the supramarginal lobule respectively.

Whenever there is present in our consciousness the sound or the visual symbol of a word, it is due to the activity of one of these centres. This activity may be excited by afferent impulses from the organs of hearing and sight, respectively; *e.g.*, the sound or sight of a familiar name excites in our mind the idea of the corresponding object; and conversely, when a known object is presented to us through the medium of our ears or eyes, its name, uttered and written, arises more or less vividly in our consciousness.

These centres may also respond to stimuli originating in the activity of other areas of cerebral structure, as when we voluntarily or involuntarily recall the sound or form of a word (associational or volitional stimuli)

[1] 'The Brain as an Organ of Mind,' pp. 601, 618.

This power of response to stimulation from an associated centre is also illustrated by the facility with which the idea of a sound is excited by the sight of its written or printed symbol, while, on the other hand, a spoken word, though directly stimulating the auditory word-centres, elicits in the visual word-centres the image of its form. This indicates a close union between the auditory or visual centres for the perception of words by fibres allowing the passage of impulses in either direction, so that each centre is capable of receiving stimuli from, or sending them to, the other.

In the expression of our thoughts in speech or writing, the action of the auditory and visual word-centres calls into activity the motor-regulating or kinæsthetic centres, by the aid of which the movements of the vocal organs in speech, and the hand in writing, are governed and co-ordinated.

The kinæsthetic centre for speech movements is, as might be supposed, more closely connected with the auditory word-centre, that for writing movements with the visual word-centre. [The kinæsthetic speech centre *may*, however, as a result of special education, be stimulated directly from the visual centre, as when deaf-mutes are taught to speak; and, similarly, the centres for writing may be stimulated from the auditory centre, as in the case of persons born blind.]

To sum up, we are possessed of an auditory perceptive word-centre capable of receiving at least three kinds of stimuli to action, viz.,

1. Volitional;
2. From ear;
3. From visual word-centre;

and capable of originating stimuli which may proceed:

1. To visual word-centre;
2. To kinæsthetic centre for speech movements.

[3. „ „ writing movements.]

We have also a visual perceptive word-centre capable of response to at least three kinds of stimuli, viz.,

1. Volitional;
2. From eye;
3. From auditory word-centre;

And which can originate stimuli proceeding to,

1. The auditory word-centre.
2. The kinæsthetic centre for writing.

[3. „ „ „ speech.]

To apply this theory to the case before us, first let us test the *auditory word-centre*.

The patient speaks spontaneously, but often forgets or misuses names. The reaction of this centre to volitional

stimuli is therefore slightly impaired, and the patient suffers from paralytic and inco-ordinate amnesia to a slight extent.

She understands perfectly what she hears. Reaction to stimuli from ear is therefore normal.

She can repeat correctly what is said to her. The relations of the auditory word-centre with the kinæsthetic centre for speech-movements are thus seen to be unimpaired.

On testing the *visual word-centre* we find that the patient can spontaneously express herself in writing, but that her written language is subject to the same slight defects as her spoken language.

Though her sight is good, written and printed matters are unintelligible to her. She is only able to recognise a letter here and there, and, with assistance, to spell out a short word. This want of reaction in the visual word-centre to stimuli from the organ of sight is further exemplified by the great difficulty which the patient experiences in naming objects presented to her view, though she is perfectly aware of their nature, and will immediately acquiesce when the right name is proposed to her.

The fact that, on handling, tasting, or smelling the objects shown her, she will often recollect their names though unable to do so on merely seeing them, illustrates the possibility of stimulating the auditory word-centre through the tactile, olfactory or gustatory perceptive centres.

That the patient can write, both spontaneously and from copy, shows that the connections of the visual word-centre with the kinæsthetic centre for hand movements are unimpaired.

Let us now turn our attention to the *commissural fibres*, connecting the auditory and visual word-centres, which are employed when one centre is called into action by stimuli directed to the other. When words are repeated to the patient she can write them down correctly. The visual centre is therefore capable of stimulation through the auditory or, in other words, the *audito-visual fibres* of the commissure are intact.

When words are written down and shown to her, or when a printed book is put before her, the patient cannot read aloud. Were her visual word-centre shown to be normal, this inability to read aloud would indicate a lesion of the visuo-auditory commissural fibres. Since her visual word-centre has been shown to be damaged, it is not improbable that the '*block*' is situated in that centre and not in the commissural fibres. It seems, therefore, that a lesion of the visuo-auditory fibres in this case is, though not incompatible with our theory, yet

hardly required by it, and, if existent, is difficult of demonstration.

It is interesting to notice the assistance afforded to the patient. in her efforts to read, by the act of passing her pencil over the letters, *as if writing them.* This phenomenon has been noticed by Dr. Bastian,[1] who quotes a case recorded by Westphal, in which it was clearly marked. "Apparently the kinæsthetic impressions" [*i.e.* impressions of "muscular sense" or "sense of motion"] "were capable of rousing related parts of the auditory word-centre"—the nervous impulses being assumed to have passed *directly* from the kinæsthetic centre for writing to the auditory word-centre or *indirectly* to it viâ the more closely related visual word-centre.

The recognition of *numerals*, while letters are unintelligible, has often been noticed in cases of this kind. Dr. Bastian[2] accounts for it by the supposition that, the individual numerals being only nine in number while the letters are twenty-six, the amount of individual attention directed to the former must have been greater, and that the patient is therefore more familiar with them. It may be urged, however, that, unless a man has been constantly engaged in pursuits which necessitate the frequent use of numerals, he will during his lifetime have devoted a much larger amount of attention to letters, since one may read or write many pages without the aid of a single numeral, while one rarely sees a page of numerals without a single letter.

I do not know of any other case in which, as in the present, it has been noticed that the vowels were better recognised than the consonants.

Note on Patient's condition, June 1887.—She is in good general health. Her memory has much improved. She can read a few lines of a printed book, but is tired by the effort, and soon becomes unable to recognise the words. Her style of reading is like that of a schoolboy translating "at sight." She goes on swimmingly for several words, then hesitates and guesses the meaning of the next word from the context. She will thus read "man" for "person," "godly" for "righteous," &c.

[1] 'The Brain as an Organ of Mind,' p. 646, note.
[2] *Loc. cit.* p. 664.

TABES DORSALIS—ATAXIA LARYNGIS.

BY WILLIAM GAY, M.D., M.R.C.P.

It is well known that affections of the larynx are occasionally found in the course of tabes dorsalis. Two of them, spasm of the adductors of the vocal cords, and bilateral abductor paralysis, have received considerable attention and are now included among the well-recognised, but somewhat rare, symptoms of the disease.

Not so, however, ataxia of the vocal cords, which, probably on account of its extreme rarity, has received but very scant notice. Indeed, the only reference to the condition I have been able to find in English or French literature is in Gowers' 'Diseases of the Nervous System,' vol. i., where it is said that "a sort of ataxic irregularity of the vocal cords has been described by Uhtoff."

I have ventured therefore, to put the following case on record, although it presents few other features of any interest:—

[1] C. A., æt. 35, coachman, came to Moorfields, September 15th, 1886, complaining of squint and diplopia. His mother is alive, æt. 70, but his father died at 60 from "drink." There is no history of any hereditary neurosis. He is married and has eight children, three of whom have died from consumption, bronchitis, and meningitis respectively. He admits gonorrhœa, but denies syphilis, of which I carefully enquired, but could find no evidence.

Twelve months ago the right eyelid drooped, the eye itself turned outwards and he suffered from giddiness and diplopia. Four months later numbness of the four fingers of the right hand came on, and he had what he called rheumatic pains down the shin, but they seem to have been of an aching character and not at all like lightning pains.

At present the right eye cannot be moved past the middle line inwards, and the upper and lower movements are limited. No nystagmus. Little ptosis now remains, but the lid is raised with a double jerk, as if two efforts were required to accomplish it. The pupils measure 3.5 mm., are equal and contract to accommodation, but not to light. Neither is round, but more or less

[1] The patient was under the care of Mr Hulke, to whose kindness I am indebted for permission to publish the case.

triangular, of course with the angles well rounded off. The near point is at seventeen inches in both eyes, indicating a considerable weakness of accommodation in a man of his age. Distant vision normal. No hypermetropia or colour blindness. Fundus normal.

Knee-jerks absent; micturition normal. Not the slightest unsteadiness with the eyes shut and feet together. No abnormality of sensation anywhere, except in the four fingers of the right hand, which are still numb. With that hand he utterly fails to perform any highly co-ordinated act, *e.g.* the buttoning of his coat. He also makes a very bad shot at his nose, when he endeavours, with eyes closed, to touch it with his forefinger. There is no such difficulty with the left hand, nor are those fingers numb. The aching pains in the legs continue. No girdle pain. Special senses normal. No bulbar symptoms. Soft palate normal. No tremor of tongue. There is occasional vomiting, but nothing at all suggestive of gastric crises. His tongue is foul, and he suffers, and has done for the last few months, from the belching of enormous quantities of wind. It happens perhaps once or twice every minute, and is evidently quite distressing. The only other patient I have ever met at all approaching him in this respect was himself a tabetic.

Twelve years ago it was noticed that C. A. had two voices, a high and a low, but what this meant I could not quite make out, nor could he explain. Twelve months ago it underwent a change and became as at present, though not so marked. It very much resembles the cracked voice of puberty. There is a rapid change occurring every three or four words from his natural, rather deep-toned voice to a high falsetto key. This peculiarity in the voice drew my attention to the larynx, where I found the following condition of affairs. There was a total loss of all rhythm and regularity in the action of the cords. They were capable of being completely adducted during phonation, and abducted during inspiration, but the latter by starts and jumps only. Sometimes they would separate for two or three mm., hesitate and then actually come together again, before being suddenly fully abducted. Theys always acted in unison, but the left cord seemed the more affected. Dr. Semon, who kindly saw the case with me afterwards, corroborated the above description, except that he did not see the cords come quite together after once they had commenced to be abducted. It is impossible to say whether this ataxic irregularity of the cords was influenced by the volitional element of respiration, or not, but my impression is that it was most marked during his earliest examinations, when he had to make greater efforts to breathe as directed, than were required afterwards.

There was no history of laryngeal crises. Thoracic respiration was carried on with perfect rhythm and regularity. Pulse 80, small, rather irregular, as if a beat were occasionally dropped. Sounds normal. Six months later his condition remained unchanged, except that his pains had disappeared and his voice was becoming more "cracked." The treatment consisted of Pot. Iod. gr. x. ter die.

The evidence in favour of the disease being primarily tabes dorsalis is not great, but I take it that the absence of knee-jerks (which was frequently tested), the presence of the Argyll-Robertson pupils, the partial paralysis of the third nerve and the numbness and inco-ordination of the fingers, are sufficient, in the absence of symptoms of general paralysis, to establish the diagnosis.

The peculiar falsetto voice may, of course, be only a coincidence and have nothing to do with the rest of the case, but I am inclined to consider it a part of the ataxia of the vocal cords, and due to a similar condition of some fibres of the phonatory muscles. If it came on, as he asserted, twelve months ago, it would correspond roughly with the commencement of the development of the disease, as shown by the third nerve paralysis and the numbness of the fingers. The condition too is evidently progressive, as he himself has noticed. Oppenheim[1] describes a case of tabes, in which there was a falsetto voice, but in this instance there were also laryngeal crises and paralysis of the posterior crico-arytænoids and right lateral crico-arytænoid muscles, as well as difficulty in swallowing and temporary failure of the heart. If we except the slight irregularity of the heart, there is nothing in my own case to point to any further affection of the 8th nerve nuclei, than that, which is probably the cause of the ataxia of the cords.

I have notes of two other cases of tabes with an irregularity of the pupils similar to that described above, so that that condition cannot be a very rare one.

[1] 'Berlin. klin. Woch.,' Jan. 26, 1885.

CASE OF MUSCULAR HYPERTONICITY.

BY ROBERT SAUNDBY, M.D. (EDIN.); F.R.C.P. (LOND.)

Physician to the General Hospital, Birmingham; Consulting Physician to the Hosp tal for Women, the Eye Hospital, &c.

JOHN MCGUIRE, aged 66, tailor, was admitted into the General Hospital on July 1st, 1887, complaining of stoppage of the bowels, weakness of the legs and arms and impaired sight. This illness began six months ago, and has gradually got worse. He noticed his sight began to fail about March, and his hands became affected at the beginning of June.

His previous health had been good; he admitted having had syphilis forty years before, and that he had been a heavy drinker. He had not met with any serious injury, fall or blow on the back.

He was a widower; had had five children, three died in infancy, two were living in good health.

He walked with his feet apart, and turned round with difficulty; he did not stand steadily with his feet together and his eyes closed, nor could he steady himself on one foot so as to step upon a chair. He had great difficulty in walking upstairs, and could only do so by holding the balustrade; after going up a few steps he became more unsteady, and there were clonic spasms in the calf muscles. He also complained of numbness and tingling in the hands; of loss of memory, especially for words, with hesitation in speaking.

There was no loss of power distinctly to be made out in any group of muscles, nor any rigidity.

There were no girdle or lightning pains; no abnormal subjective feelings of heat or cold or formication; no impairment of sensibility to touch, tickling or pain. Pupil reflexes normal. Knee-jerk exaggerated on both sides. Superficial reflexes present; no ankle-clonus. Ophthalmoscopic appearance of fundus in both eyes quite normal, except some natural presbyopia.

Progress of Case.—By July 5th he could walk alone fairly fast and with confidence. The gait was jerky and he picked up his feet too high. The clonus of calf-muscles had gone.

July 6th.—Articulated quite well, and said that he had

lost the numbness in his fingers. On July 11th he said the difficulty of speaking had "gone away from him," that he could remember better what he read. He continued to progress till his discharge on July 20th, when he left, able to walk very well.

When this case came under my observation, I knew nothing of the group of cases described so well by Dr. A. Hughes Bennett at the Neurological Society last summer. As it appears to be an illustration of a rather rare condition, and one that puzzled me a good deal at the time, I venture to send these brief notes for publication. The treatment used was the daily inunction of a drachm of mercurial ointment, ten grains of iodide of potassium three times a day, and a draught containing fifteen minims of fluid extract of cascara at bedtime when the bowels were not open. I do not now attach any importance to the specific treatment in the cure that resulted, but attribute this chiefly, if not altogether, to the rest.[1]

[1] Since this was written he has been an in-patient again, and the result has a little modified my opinion as to the effect of specific treatment; for he seemed to make very little improvement until the mercurial inunctions were resumed. He is now (Dec. 6th) fairly steady on his legs, and the knee-jerks are less exaggerated.

A CASE OF EPILEPSY WITH EXOPHTHALMIC GOITRE—NEUROTIC HISTORY.

BY JAMES OLIVER, M.B., F.R.S. (EDIN.)

Rosa S. æt. 23, single, came under my care, complaining of swelling in the neck, palpitation, and prominence of the eyeballs. Menstruation, established at the age of seventeen, has been regular in its recurrence till twelve months ago, since when there has been complete amenorrhœa. The monthly flow has always been scanty, frequently amounting only to a few drops.

At the age of seven, the patient began to suffer from epileptic disturbance, originating in, and being confined to the higher intellectual nerve centres, determining merely a loss of senses. During such attacks she would maintain her position throughout, and although talking or engaged in any amusement, would as a rule evince nothing more than a peculiar, yet withal characteristic stare and fixity of the eyes. Occasionally when this abolition of consciousness was somewhat prolonged, she would partially undress herself, a disposition unfortunately too often manifested by patients during this epileptic state. From seven till seventeen years of age, when the functional activity of the uterus was manifested, the epileptic attack of almost daily occurrence had consisted merely of fleeting unconsciousness, spoken of by the patient as a "blank." With the initial establishment of the catamenial discharge, however, she was the subject of her first fit, or motor manifestation of the epileptic disturbance, the discharge which hitherto had been confined to the higher nerve centres, having spread to and invaded all the cerebral representative structures of the body. It is frequently alleged that females who suffer from epilepsy in early life will, on attaining the age of puberty, when the generative functions become evolved, experience immunity from this grave nerve-disorder. Although this may very occasionally happen, close scrutiny teaches us that this age corresponds with that recognised as the greatest epileptogene period, and that the molecular nerve disturbance is more likely to be augmented than lessened, because of a defective power of adaptation on the part of the epileptic individual to those changes correlated with the establishment of sexual activity.

A year ago, patient began to complain of feeling languid and tired, the slightest exertion induced shortness of breath and a sharp sudden pain referred to the pit of the stomach. Two months later, she remarked the existence of a slight swelling in the neck, which has progressively increased, and is the result of change in the thyroid gland, affecting more especially the left half of this structure. Simultaneously with the appearance of thyroid enlargement, a prominence of the eyeballs was noted, which too has been slowly progressive. Occasionally an eruption appears in the front of both legs, of the nature of erythema nodosum. The pulse numbers 120 per minute, whilst the temperature registers 99°·4 F. There is no cardiac bruit to be detected.

Family history.—The patient's paternal grandfather was addicted to alcohol, and died at the age of twenty-nine from some pulmonary disease. The father and mother are both living: the father, aged fifty, is epileptic, and has been subject to fits ever since he was eighteen years of age; the mother is apparently healthy. One brother died at the age of ten, after an illness of three weeks, the symptoms being those of a cerebral character. Another brother, aged eleven, is at present under my care. He is totally blind, suffers from severe headache and occasional sickness. The history given by the mother is that the boy four years ago had a fall at school, after which he complained of headache for one week only, and remained apparently perfectly well till twelve months ago. The mother then noted that every morning after a night's rest, on leaving the recumbent position, he was sick. At the same time he began to suffer from occasional violent pains in the head, which caused the little fellow to scream out. Two months later he was noticed to stumble over little things which lay in his way, and also that he could not distinguish articles of food on the table. The loss of vision has been slowly progressive, so that now he is wholly unable to appreciate a strong light reflected from the mirror when it impinges on the retina. There is marked atrophy of both optic discs, which present a somewhat pearly aspect with enlarged veins. The symptoms are those of a new growth in the brain, but there is nothing to guide us in determining its location.

ATAXIA IN THE DONKEY.

NOTE BY DAVID DRUMMOND, M.D.

While spending a few days at Moffat in Dumfriesshire, in company with a small party of friends, amongst whom was Dr. Rutherford Morison of Hartlepool, an interesting case suggesting important questions in comparative pathology came under our notice. The subject of our observations was a donkey of uncertain years, though I gathered that he was considered in the neighbourhood to have passed the ripe age of twenty. My attention was first called to his condition by an enterprising young lady of an inquiring turn of mind, who discovered the animal's peculiarities on inducing him to follow her, which he attempted to do with considerable alacrity, displaying at the same time a most singular gait, characterised by the grossest inco-ordination, amounting almost to the grotesque. He reeled from side to side, and staggered along, throwing his legs out in a most irregular manner. Occasionally he seemed on the point of falling, but always managed to save himself. The impairment appeared to be due more to a want of control than to actual feebleness, for the shifting of the feet necessary for the maintenance of his equilibrium in standing or walking was performed in a fairly active way.

Being much interested in the case, and having found the owner's cottage, Dr. Morison returned with me next day to make further observations. We found that the pupils were mere pinholes and did not respond to any form of stimulus. It was apparent that the gait resembled closely the mode of progression of many tabetic patients, and the resemblance to locomotor ataxia was intensified by the discovery, that the poor animal was quite unable to walk or stand with his eyes blindfolded, for on being tested in this way, he reeled for a moment and then fell to the ground.

Having effected a purchase, and laid under contribution the services of the neighbouring gamekeeper, a *post-mortem* was made by the aid of some instruments kindly lent by Dr. Grange of Moffat, and the lumbar enlargement of the spinal cord and part of the great sciatic nerve were removed for

microscopical examination. Examined fresh, the posterior columns were entirely free from the appearances so characteristic of the ribbon sclerosis of tabes.

After hardening, the sections of the cord, stained with carmine, showed a deep red zone extending nearly half-way round the postero-lateral surface. On microscopical examination, the deeper stained area was seen to be an annular or marginal myelitis or sclerosis. It embraced both posterior roots and extended into one lateral column. The changes were evidently chronic, and appeared to be independent of meningitis. They were essentially interstitial; and little else than a dense overgrowth of neuroglia, in which were a large number of nuclei and a few nerve elements, could be seen. The remainder of the spinal cord was healthy, and the sciatic nerve was normal.

Newcastle-on-Tyne.

CASE OF CANCER OF THE RIGHT LOBE OF THE CEREBELLUM AND LEFT LENTICULAR NUCLEUS; MARKED VERTIGO; NO PARALYSIS.

BY BYROM BRAMWELL, M.D., F.R.C.P. (EDIN.)

OBSERVERS are pretty well agreed that lesions of the corpus striatum, so long as they do not implicate the motor strands of the internal capsule, may be unattended with any marked or persistent paralysis.

In the 'Edinburgh Medical Journal' for February 1879, p. 697, I have repeated a case in which almost the whole of the left nucleus caudatus was destroyed by a tubercular tumour, and in which there was no localised loss of motor power. In the following case, in which a secondary nodule of cancer had practically destroyed the whole of the left lenticular nucleus, the mass of new growth being very sharply defined and limited to the ganglion, paralysis was also absent.

The very marked character of the vertigo and the peculiar sensation which the patient experienced of "the ceiling falling down on the top of her" are also interesting features of the case, and were doubtless due to the large nodule of cancer which has developed in the right lobe of the cerebellum, and which was exerting marked pressure upon its middle peduncle.

The development of the new growth within the cranium was somewhat slow, for death did not take place until six months after the cerebral symptoms declared themselves.

The notes of the case are as follows:—

Mrs. A., æt. 61, was seen in consultation with Dr. Murray of Galashiels, on 13th July, 1886.

Eight months previously Dr. Murray had excised the left mamma and some of the axillary glands, which were affected with cancer. The patient made a satisfactory recovery, the wound healing by the first intention; and remained in good health, for six months—until the end of May, 1886. She then began to suffer from severe headache and vertigo; her gait became unsteady, and like that of a person affected with drink.

On *June 1st*, she was seized with what her daughter thought

was a fit. Dr. Murray, who was immediately sent for, found her sensible, but somewhat confused; the muscles of the right side of the face were paralysed, the mouth being slightly drawn to the left. She complained of "dreadful" pains all over her head. She remained in much the same condition until June 20th, when she had another so-called fit or faint. Dr. Murray was unable to satisfy himself that there were any distinct spasmodic muscular contractions; the attack, he says, resembled rather a faint than an epileptic fit. After this attack, the patient became much excited, like one suffering from delirium tremens: she insisted upon getting out of bed; wanted to dance; said she felt fine, &c. After a week's duration, this excitement subsided, and was succeeded by vomiting.

At the time of my visit, the headache, vomiting, and vertigo were said to be very persistent and severe. The patient complained that, when her eyes were open, she felt as if the ceiling were falling down on the top of her, and that objects in the room were moving round her; she was unable to say in which direction they moved. The retinal veins were large, and the edges of the disc swollen and blurred; in other words, there was distinct, but not intense, double optic neuritis. There was no perceptible paralysis in any part of the body.

The glands on the left side of the neck were enlarged and evidently cancerous; and it was evident that secondary deposits of cancer were also developing within the cavity of the cranium—in all probability in the cerebellum.

Subsequent progress of the Case.—For some three months after my visit, the patient remained much *in statu quo*, the headache, vomiting and vertigo being very severe and distressing. Towards the later stages of the case, the severity of the symptoms abated, the mental faculties became clouded; and for the last month of her life the patient lay in a semi-comatose condition. After the first fit, the right side of the face was, as has already been mentioned, temporarily paralysed; but the loss of power disappeared after a couple of days. Throughout the whole course of the case, Dr. Murray was unable to detect paralysis in any other part of the body. It is of course impossible to assert that there may not have been some paralysis in the later stages of the case—when the patient lay in a semi-comatose condition; but if any localised loss of motor power was present, it was not recognisable. Dr. Murray assures me in the most positive manner that (with the exception of the temporary loss of power in the facial muscles, which has been referred to above) there was never, so far as he could discover, any paralysis. I may perhaps add that the case was carefully watched, for both Dr. Murray and myself were much interested in the peculiar vertiginous sensations; and Dr. Murray kindly undertook to note any fresh symptoms, which might develop after my visit, and to obtain permission, when the patient died, to examine the body *post-mortem*.

Death took place on November 25th; and I made the autopsy on November 27th, the head only being examined.

There were extensive deposits of cancer in the cicatrix, left axilla, and left side of the neck.

The scalp, skull-cap, and external surface of the dura mater were normal. The convolutions were somewhat flattened, but the surface of the brain was by no means so anæmic as one often sees it, in cases of intra-cranial tumour. The arteries at the base of the brain were markedly atheromatous.

The right lobe of the cerebellum was adherent to the dura mater; and a firm hard mass could be felt in its interior. There were no deposits of new growth to be seen on the surface of the cerebrum.

The organ was injected with Müller's fluid, and hardened, as a whole, in the manner described on page 435.

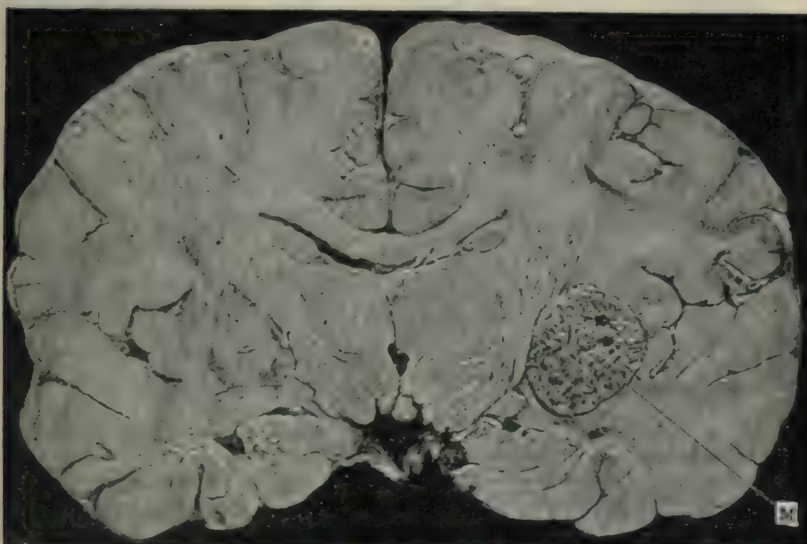


FIG. 1.—Transverse vertical section through the brain of Mrs. A., showing a cancerous deposit (M) in the position of the left lenticular nucleus. Considerably less than the natural size.

At the end of six weeks it was cut into a series of transverse vertical sections. A small nodule of cancer, about the size of a three-penny piece, was present in the upper end of the right ascending parietal convolution. A larger nodule, on section about the size of a shilling, had completely destroyed the lenticular nucleus on the left side (see Fig. 1). The mass of new growth was sharply defined and singularly limited to the position of the ganglion, the adjacent strands of the internal capsule being apparently little, if at all, implicated. A large mass of cancer, fully the size of a small hen's egg, was situated in the right lobe of the cerebellum; the

adjacent middle peduncle was considerably compressed by the mass of new growth.

On microscopical examination, the structure of these tumours was found to be typically cancerous.

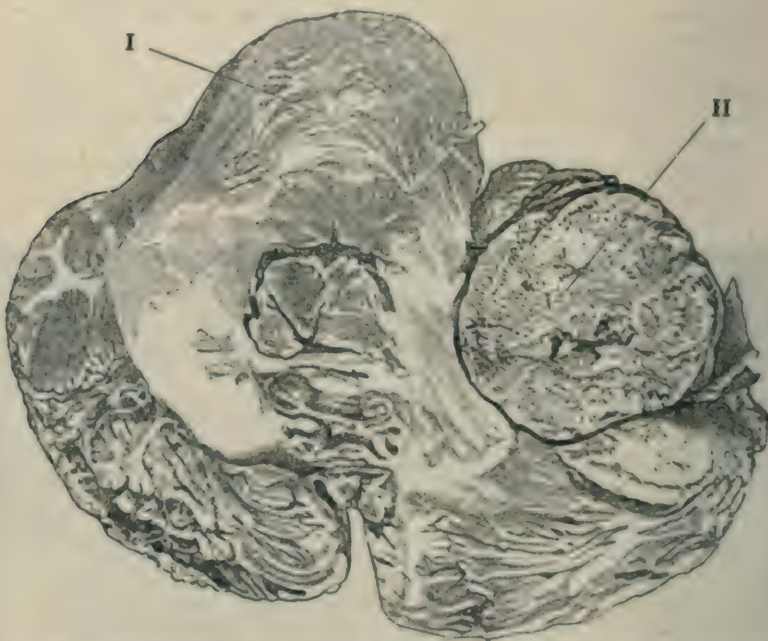


FIG. 2.—Transverse section through the pons Varolii and cerebellum, showing a large cancerous mass in the right lobe of the cerebellum. Somewhat less than the actual size. The letter I points to the pons Varolii, and II to the cancerous tumour.

A PECULIAR CASE OF LEAD-POISONING IN WHICH THERE WAS MARKED LOSS OF VISION, BOTH FOR WHITE AND FOR COLOURS, WITHOUT ANY CHANGES IN THE FUNDUS OCULI; AND IN WHICH RAPID RECOVERY TOOK PLACE UNDER SULPHATE OF MAGNESIA AND IODIDE OF POTASSIUM.

BY BYROM BRAMWELL, M.D., F.R.C.P. (EDIN.)

In the great majority of cases of lead encephalopathy, in which dimness of vision is a prominent symptom, well-marked changes in the optic discs—double optic neuritis or optic atrophy—are recognisable by means of the ophthalmoscope.

The following case is, in my experience, quite exceptional, inasmuch as the discs were absolutely healthy.

The rapid manner in which the symptoms disappeared under treatment; the peculiar character of the limitation of the colour fields; and the presence of the muscular tremor—a symptom which is amongst the rarer manifestations of lead-poisoning—are interesting and important features of the case.

The notes of the case are as follows:—

J. H., æt. 30, employed in a paint manufactory, came under my notice as an out-patient at the Edinburgh Royal Infirmary, on October 19, 1887, complaining of dimness of vision, headache, general malaise, and exhaustion.

Previous History.—The patient stated that, until three years ago, he was employed as a fireman on board a steamer; and that until a year ago, when his present illness commenced, he enjoyed excellent health. He has never suffered from syphilis or gonorrhœa.

For the past three years he has worked in a paint-mill, and has had to take his turn with the other men in "mixing the litharge." Knowing that many of the men employed in this way suffer from lead-poisoning, he has been on his guard against it, and has been scrupulously careful to brush his teeth and wash out his mouth on returning home from work.

Twelve months ago, he first began to feel ill; and he has never been well since. During this period, he has lost colour, and has daily suffered from headache; the pain, which is chiefly frontal and worse at night, has at times been very severe; to use his own words, "it is sometimes awful." He has also suffered much

from sleeplessness. The bowels have been obstinately constipated, and he has had severe pains in the abdomen. The headache and sleeplessness were, he says, for the time relieved by purgative medicines. The abdominal pain, unlike that in most cases of lead colic, is, he says, increased rather than relieved by pressure.

For six months he has suffered from a constant trembling in the arms and legs, more especially in the right hand and arm.

Four weeks ago, his eyesight began to fail, and he can now hardly see at all. A fortnight ago he was able to read the newspaper. He states that he has occasionally seen double.

His appetite is bad; he feels very ill and weak; and has lost a great deal of flesh.

He has never vomited, but when working in the litharge he used to feel very sick and inclined to vomit. There has been no vertigo.

Present Condition.—On examination, the patient was seen to be very pale and anæmic. A small, swollen, projecting portion of gum, situated between two of the incisors in the lower jaw, presents the blue stain characteristic of lead poisoning; but the other parts of the gum are quite healthy. The central incisors in the upper jaw are wanting; the teeth are for the most part clean, and look as if they had been well cared for.

Vision is markedly impaired. In the right eye V. = less than $\frac{20}{200}$; in the left eye V. = rather more than $\frac{20}{120}$. Colour vision is much impaired, greens being confounded with blues, dark blue is said to be light blue, and green is said to be a darker blue than dark blue.

The fields, both for white and for colours, are very much constricted in both eyes—a little more so in the right than in the left. In Fig. 1, the condition of the field for white is represented as it was on October 19th; and the fields for colours as they were on October 27th (*i.e.*, when vision had already considerably improved).

In Fig. 2 (p. 510) the condition of the fields, both for white and for colours, is shown as they were on November 15th.

By reference to Fig. 1, it will be seen that the constriction of the yellow field is proportionately much greater than the constriction of the fields for green, blue, and red. The red field seems to have suffered least.

On November 15th, the colour fields were practically normal, with perhaps the exception of a slight degree of constriction towards the right.

The pupils are equal, and moderately dilated; they contract, though not perhaps as briskly as normal, both to light and accommodation.

The fundus in each eye is absolutely healthy. (Dr. Berry, who was kind enough to examine the case, corroborated the foregoing statements as regards the condition of the fundus and the state of vision. His report was briefly as follows:—"I find V. = [illegible] in both eyes; the fields concentrically limited; colour vision abnormal, especially defective for yellow and blue; blue and green

confused. Ophthalmoscopically, nothing abnormal. History of vision having become recently much worse. *Diagnosis.*—Poisoning of visual centres? I think this more likely than that there is any lesion elsewhere, chiefly on account of the colour defect, which is unlike anything else but hysterical amblyopia.")

There is slight nystagmus on moving the eyes in the lateral direction; and there seems to be some slight paralysis of each external rectus muscle.

The right hand and arm, and to a less degree the left hand and

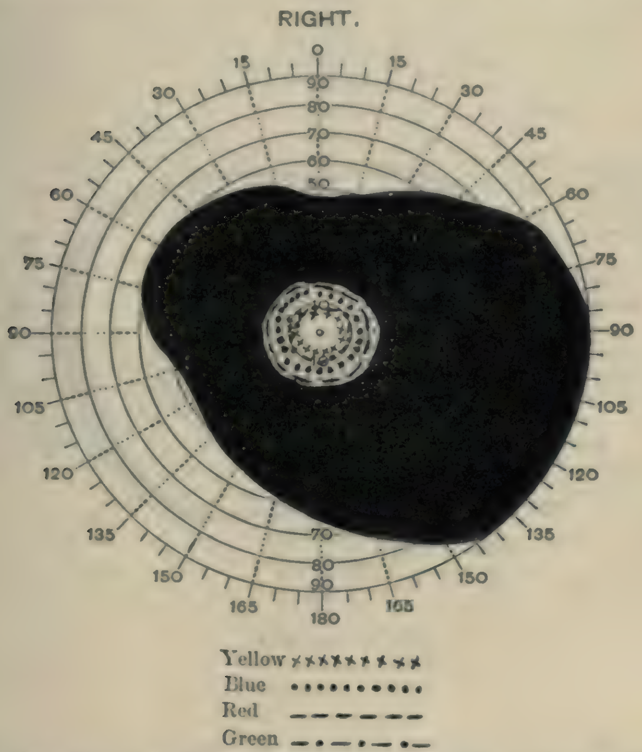


FIG. 1.—PERIMETER CHART OF THE FIELDS FOR WHITE AND FOR COLOURS IN THE RIGHT EYE, SHOWING MARKED CONTRACTION.

The field for white was mapped out on October 19th; and that for colours on October 27th (when considerable improvement had already taken place).

both lower extremities, are the seat of a fine tremor, which seems to be independent of voluntary movement or external irritation. There is no localised loss of motor power, and in particular no wrist-drop or localised paralysis in the extensor muscles of the forearms; the grasping power of the hands, as measured by the dynamometer, is equal (105).

The knee-jerk is exaggerated in both legs, more especially in the right. The superficial reflexes are normal.

Tactile sensibility is normal. The urine, which was examined on several occasions, was found to be normal; the presence of albumen was never detected. Dr. Stevenson Macadam, who was so good as to analyse one specimen for me, was unable to detect the presence of lead.

Diagnosis.—The case was evidently an exceptional one, but

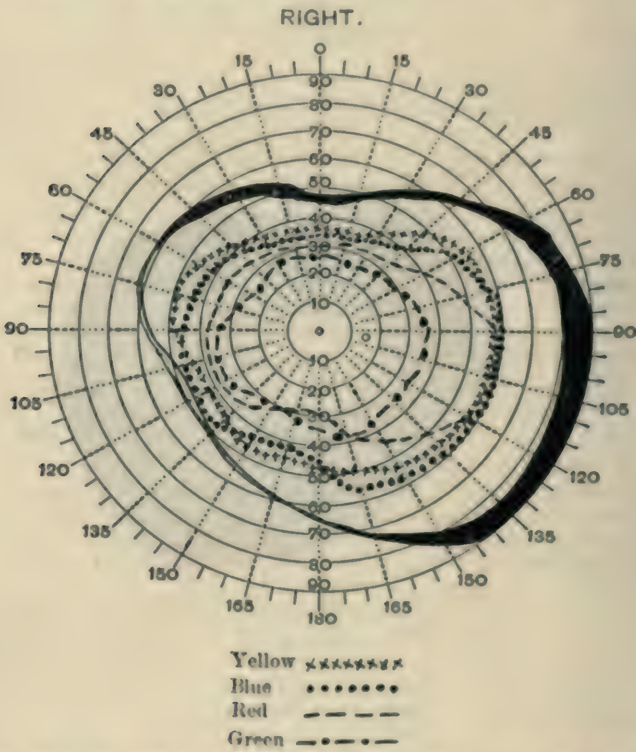


FIG. 2.—PERIMETER CHART OF THE FIELDS FOR WHITE AND FOR COLOURS IN THE RIGHT EYE, SHOWING THE CONDITION AFTER RECOVERY (NOV. 15th).

The fields are practically normal; the extent to which the white field is still contracted is represented by the dark area.

the opinion formed was that the symptoms were due to lead-poisoning.

The occupation of the patient (which exposed him to the risk of lead-poisoning), the constipation, the blue patch in the gum, the colic, together with the fine muscular tremor, were in favour of plumbism rather than of intra-cranial tremor, which also suggested itself as a possible cause of the symptoms. The fact that there

was neither optic neuritis, vomiting, nor vertigo (when taken in conjunction with the positive facts in favour of lead-poisoning which have been mentioned above) seemed to exclude intra-cranial tumour.

The striking manner in which all the symptoms disappeared under the administration of large doses of iodide of potassium and sulphate of magnesia, corroborated the diagnosis of lead-poisoning.

It is strange that the chemical analysis failed to show the presence of lead in the urine; but this fact, *per se*, is hardly, I think, sufficient to destroy the diagnosis and to counterbalance all the other facts in favour of plumbism.

Treatment and Progress of the Case.—Sulphate of magnesia and iodide of potassium were prescribed in full doses. Under the treatment, rapid improvement took place.

On October 25th, when Dr. Berry examined the patient, vision had already improved.

On November 1st, the patient was looking altogether better and brighter. He stated that he was free from headache, and that he was sleeping and eating well, and feeling very much stronger. The muscular tremor was now scarcely perceptible. The acuity of vision, both for white and for colours, was normal; and the constriction of the fields, both for white and for colours, had almost entirely disappeared.

On November 15th, the fields were carefully mapped out by means of the perimeter, and were found to be practically normal (see Fig. 2). The muscular tremor could no longer be detected. The patient was entirely free from headache, and expressed himself as feeling very well.

On December 11th, the patient stated that he had been entirely free from any symptoms of lead-poisoning since the date of the last note. He is anxious to return to work, and would have already done so, had he not had a slight bronchitic cold, and had he not been afraid of again suffering from lead-poisoning.

Reviews and Notices of Books.

The Brain considered Anatomically, Physiologically, and Philosophically. By EMANUEL SWEDENBORG: Edited, Translated and Annotated by R. L. Tafel, A.M., M.D., Ph. D. London: James Speirs, 36, Bloomsbury Street. 1887.

IN vol. ii. of this work the editor reproduces in successive chapters Swedenborg's views on the pineal gland, the third ventricle, the infundibulum, the pituitary gland, the cavernous sinuses, including the smaller sinuses in this situation, the rete mirabile, the cerebellum, the medulla oblongata, the fourth ventricle, the pons, and the corpora pyramidalia and olivaria. The plan followed in the book is first to give Swedenborg's own description along with that of the authorities quoted by him. From these quotations we see that Swedenborg, although not setting himself up as an anatomical authority, had nevertheless carefully examined the structure of the human brain, and had also to some extent compared its structure with that of some of the lower animals; but he unquestionably establishes a claim on our consideration by the familiarity he shows with the descriptions and views of the best anatomists of his day. On the structure of the pituitary gland, for instance, he quotes Willis (1664), Vieussens (1685), Ridley (1695), Heister (1717), Littre (1707), Bianchi (1715), Winslow (1732), and Morgagni (1740). As his own statements regarding the facts of structure do not differ materially from those authorities, it is impossible to set aside what he says as visionary, although of course the inferences he draws from recognised facts of structure may or may not have been correct. The editor in his preface takes to task Dr. Maudsley for his statement that, in 1743 and 1744 (at the time, that is, when he was engaged on the work on the Brain at present under review), Swedenborg was "subject to seizures which were closely akin to, if they were not actually, epilepsy." Dr. Maudsley also says that in 1743 Swedenborg had "an attack of acute mania," and that "when the acute attack passed away, a monomania was left behind, which was the morbid evolution of his self-sufficient character." Dr. Tafel repudiates and contradicts this, saying that when Swedenborg returned to his native country in 1745, no one noticed anything of the monomania; that from 1745 to 1747 he again attended to his duties as Assessor at the College of Mines; where he acted both in the character of a public administrator of

mines, and as a judge in disputed cases; that in 1747 the college unanimously recommended him for a vacant councillorship of mines, which Swedenborg declined; and that in consideration of his long and faithful services the King of Sweden allowed him from that time till his death half of his salary annually. Even if therefore Swedenborg had some attack, such as Dr. Maudsley asserts, but which Dr. Tafel wholly denies, it is plain it must have passed off before very long.

In any case we are compelled to examine entirely on its merits the work done by Swedenborg on the Brain. The quotations from recognised anatomical authorities certainly bear no trace of garbling or unfair contortion from their plain straightforward meaning; while his reasonings from the facts before him must be examined as those of any other man who states his views, and the evidence on which he founds them. In order to put the reader in possession of all the facts regarding each part, the editor then, after quoting the authors referred to by Swedenborg, introduces the views of modern authors by quotations from their works; and it ought here to be said that the number of such authors quoted is very considerable, involving a great deal of labour on the part of the editor, so much so indeed that it is more than doubtful whether this book is not the most valuable work, even as bearing only on the descriptive anatomy of the Brain, which has ever appeared. The editor is certainly to be congratulated, both on his industry and on the skill with which he has brought together the descriptions and views of the various authorities upon the subject; his extensive linguistic attainments rendering him able to do this in a manner that comparatively few could imitate. The list of books drawn upon, *e.g.*, in the preparation of the editor's sixth note on the 'Pituitary Gland and the Sinuses of the Sella Turcica,' covers five pages, and refers to no fewer than 46 works on the Pituitary Gland, and to 29 on the Sinuses (some of the works, of course, bearing on both subjects). As to the accuracy of these quotations, we are perhaps scarcely competent to make any statements, many of the works not being accessible to us; but we may say that, having verified the quotations in a considerable number of cases, we have invariably found them correct.

After the quotations from modern authorities, which are of course the editor's own special work, he next proceeds to introduce us, in the case of each part, to what Swedenborg called his 'Analysis'—otherwise a statement of what he believed to be the final purpose or end for which each part exists, and the function which it fulfils. It is here of course that the views and reasonings of the author are specially brought out; and it is here, if anywhere, that opinions will differ as to whether his views are correct, and as to the question therefore whether he has or has not made out his case. His opinions certainly, or at least some of them, strike the medical man as novel. His general view of the function of the brain, for example, was that it is threefold. It has first the function of sensating and perceiving; second, that of

determining and acting; and third, that of conceiving and bringing forth the nervous fluid or animal spirit, *and the blood.*

Dr. Tafel truly says that modern science is acquainted with only the first two of these functions; of the last it knows nothing. Is this novel function a real function? What evidence is there that the whole idea of it is not a vision of Swedenborg's imagination? In order to obtain an answer to this question we beg the reader to read and study Dr. Tafel's elaborate Notes III. (vol. i.) and VI. (vol. ii.). He will there find the complete statement of Swedenborg's views, which he can then compare with his idea regarding the cerebellum and the cerebro-spinal fluid (Note VII., vol. ii.). We can promise him that, whatever conclusion he may arrive at regarding the soundness or otherwise of Swedenborg's opinions on these questions, his own horizon will have been considerably enlarged, his information extended, and the data on which to form an opinion of his own very considerably increased. It is almost impossible in a review to sum up the evidence adduced by the editor in favour of Swedenborg's views, because to shorten it is nearly always to weaken it. But it seems desirable to at least indicate some of the important points; and this we attempt to do, in order to draw attention to the author's views, and to the kind of evidence by which his editor supports them; but begging the reader who may be in any difficulty to refer to the notes themselves for fuller exposition. According to Swedenborg, the pituitary gland is "the last of the organs of the whole chemical laboratory of the brain." "It needs must be," he says, "that the brain has in view and carries out here some sublime and grand work which concerns the whole kingdom, and on which its welfare depends." Ecker (1853) and Sapolini (1879) declare that they know nothing about the functions of the pituitary gland; they speak of it as an "enigmatical organ," and as "a myth." Yet, on the other hand, no less an authority than Virchow says (1857) it "is almost the most constant of formations in the whole series of vertebrata"; and W. Müller (1871) expresses himself to the same effect. It certainly stands to reason that an organ so constant must have an important office. What is that office? Leydig (1852) after describing its situation and connections in the Raja clavata, says: "The closed sacs, of which it consists, throughout the whole extent of the gland, are encircled by very many blood-vessels, and what appears strange, in the lateral productions of the gland, which abound with vessels, and chiefly there, *are found blood-globules in their various retrograde metamorphoses.*" And more to the same effect: from which we may conclude that Leydig thought the pituitary gland reduces the red-blood corpuscles into fragments, and that fragments of these red corpuscles are developed into white or lymphatic blood-globules. Shortly, then, Leydig looks upon as a blood-gland that pituitary body which Ecker and Sapolini declare to be "an enigma" as to its functions. There is no reason to think that Leydig had read Swedenborg; yet Swedenborg had said, 109 years before Leydig wrote, that the pituitary body is a blood-gland. This is surely very remarkable.

But not only did Swedenborg say this, but he said it in a most elaborate and most minute manner, going into great detail as regards the structure of the gland, as to its blood-supply, as to its relations to the sinuses in its neighbourhood, and as to the mode in which it performs its functions. As regards the last point indeed, we are not sure that we are able to follow him in his account of the three orders of fluid of which the blood is composed; and we are still in doubt whether he has not made an unnecessary distinction between the animal spirit, or fluid of the first order, which seems according to him to be expressed from the fibre of the medullary substance of the brain, and the spirituous lymph, which appears to come from the lateral ventricles and choroid plexuses.

Dr. Tafel, it is true, pays much attention to this distinction, appearing thoroughly to accept it, and even writing special notes upon each of the two kinds of fluid; but after careful study of all he has said, we confess ourselves unable thoroughly to distinguish between the two. Of the second sort of fluid indeed there are said to be two kinds: "*first*, that which in the infundibulum is secreted from the ventricular fluid, and which, when properly equalised, that is, furnished with a sufficient amount of refined serum as well as of animal spirit, is discharged in that form from the pituitary gland; and *secondly*, that same fluid which, in the posterior lobe of the gland, is surcharged with animal spirit, and acts thus as the bearer of the fluid of the *first* order, from the pituitary gland." It is quite possible that the reception of the idea, that the pituitary gland is a blood-gland, has not had time to assimilate itself in our minds; but we certainly fail to obtain a clear idea of what is meant by the above quoted passage and others like it. According to Swedenborg the fluid of the third order is a grosser serum, which when supplemented by the chyle from the thoracic duct is instrumental in preparing the more composite red blood out of the former more spirituous ingredients.

Swedenborg's view of the pituitary body as a blood-gland may be summed up in his own words, that it "is a place of exchange, a mart, where the medullary substance of the brain meets the tissues and vessels of the body, and where the spirit of the brain is finally prepared for a marriage with the lower essences of the body. All the powers of the brain" (why *all?* since neither the function of sensating and perceiving, nor yet that of determining and acting can possibly be affected by the pituitary gland), "all the powers of the brain thus concentre in the pituitary gland, and through its mediation the riches of the higher sphere of the brain are communicated to the lower sphere of the body." This language, it will be observed, is scarcely scientific: it is imaginative and fanciful, rather. No doubt there is a scientific substratum; but we imagine that it must be greatly on account of the fanciful terms in which his reasoning was clothed, that the views of Swedenborg have not been more widely studied. To say that the fluid expressed from the medullary fibres of the cerebrum, and passing through the lateral ventricles into the third ventricle,

and thence into the pituitary gland (some of the fluid passing outside the gland, and some through the infundibulum), to say that this fluid is carried into the blood of the sinuses at the base of the cranium, where it helps to keep fluid that blood; and to say that the pituitary gland breaks up the fluids that pass into it—these would be plain statements of what Swedenborg believed to be facts, and of what actually appear, in the light of further experience and investigation, to *be* facts. But when such processes are called a "marriage of the higher with the lower essences," scientific minds turn away from the statement. At the same time allowance must be made for the mode of expression in vogue in his day; and it may be freely admitted that the truth or otherwise of the alleged facts is of far more consequence to us than the language in which he stated them.

Swedenborg's remarks about "marriage" of the higher with the lower essences imply, that the pituitary gland must be composed of brain matter on the one hand, and of ordinary tissue on the other. Morgagni, in Swedenborg's days, taught that the gland was composed of two different kinds of tissue; but he does not seem to have been aware how it was formed, and how, as we now know, the posterior part of it is a cerebral outgrowth (hypophysis cerebri), while the anterior part is formed by a diverticulum from the buccal epiblast; and again it has to be admitted that Swedenborg's prevision on this point was most philosophical and most sagacious. Nay, more. There is a good deal of difference of opinion among anatomists as to the microscopic structure of the pituitary gland, some of them saying that there are nervous elements in the composition of the posterior part, and some that there are not.

Luschka (1860) and Heule (1873) say that the nervous elements of the posterior part of the gland are in a state of apparent dissolution and disintegration; and it is certainly most remarkable that we find Swedenborg saying in 1743 "that fibre, namely, the cerebral fibre in the posterior lobe, does not again leave the gland, but it terminates there, and deposits its spirit there." Luschka again declares that these peculiar nervous fibres are met with chiefly in the circumference of the posterior lobe; while Swedenborg says, "These fibres discharge their animal spirit nowhere else than in the outermost parts and circumferences of the pituitary gland, where the muscular fibre and an abundant astery prevails."

To continue the account of the three fluids according to Swedenborg; we find him saying that the fluid of the *first* order secreted in the infundibulum and in the pituitary gland is carried into the inferior transverse sinus of Littre and the superior circular sinus of Ridley. From these he says it is infused into the superior petrosal sinuses, and thence into the jugular veins. Modern investigation has however shown, that the inferior transverse sinus empties into the cavernous and not into the superior petrosal sinus; so that in this point Swedenborg was wrong.

With this exception however his statement is quite in accord with the latest investigations. The fluid of the *second* order, according to Swedenborg, which is simply strained off from the ventricular liquid, and collects in the central cavity of the pituitary gland, passes into the interior of the sphenoid bone, where he says it is thoroughly mixed with arterial blood from the carotid arteries. But he did not quite know what became of it afterwards. But modern investigation has settled this in part; and at the very time when Swedenborg was writing, in 1743, an Italian anatomist was in the act of discovering a bony canal conveying venous blood, and which is now known (or is it yet generally known?) as the canal of Tabarrani. The discoverer described it minutely as lying underneath the groove of the inferior petrosal sinus, between the basilar process of the occipital bone and the petrous portion of the temporal bone, "exactly there where a certain substance is found which is almost intermediate between cartilage and ligament." The beginning of the sinus is found between the carotid and the cella turcica and its end in the foramen jugulare in connection with the inferior petrosal sinus. The history of this canal is curious. Keiller described it on the authority of Tabarrani, and it was known to Malacarne and Bell. Nevertheless so completely did it drop out of knowledge that it had to be re-discovered by Dr. Englisch of Vienna in 1863, just 120 years after its original description by Tabarrani. Nay, further, it has actually, since, been re-discovered by Trolard in 1870, he being evidently unacquainted even with the work of 1863, much more with that of 1743. This is not by any means the sole example of things becoming known and then again forgotten, nor of the waste of labour which so often occurs in the history of science; but it brings into strong relief Swedenborg's insight into the circulation of the brain, that this canal when it is discovered should exactly fit in with what is required to convey away from the interior of the bone, what he calls the fluid of the *second* order. The other part of Swedenborg's statement, that the fluid of the *second* order passes into the sphenoid bone, is confirmed by Langer (1885), Henle (1871), Londzers (1868), and Müller (1871), who all find foramina on the surface of the bone leading to its interior, though perhaps it would be unfair to say that all these authorities accept Swedenborg's views as to the kind of material which is being conveyed in the bone.

Swedenborg's fluid of the *third* order is the residuum of the ventricular liquid after the spirituous lymph—the fluid of the *second* order—has been drained off from it in the infundibulum. He says, "It is composed of the lymph of the choroid plexuses, which also seems to be charged with nervous juice—cerebro-spinal liquid." Through special tubes or pores this fluid is conveyed from the infundibulum into the posterior part of the gland, where it collects on the surface of the gland under a production of the dura mater. And during the expansion of the gland this serum is discharged into the cavernous sinuses through an intercavernous sinus or vein which runs along the lower posterior surface of the gland.

According to Dr. Tafel, the Brothers Wenzel (1812), and Sapolini (1879), prove incontestably that there is a direct line of communication for a grosser fluid between the posterior surface of the gland and the third ventricle. Peremeschko also agrees with this view. Swedenborg says this liquid of the *third* order is excreted in the posterior part of the gland, and Bianchi as well as Morgagni both admit the existence of a receptacle in this situation.

These views of Swedenborg imply certain statements regarding the course and functions of certain of the sinuses of the cerebrum, some of which agree with those of anatomical authorities, and some not. Into these statements Dr. Tafel enters very carefully. One would have thought, regarding the course taken by the cerebral sinuses, there would not be any dispute among anatomists. It is, however, rather surprising to find different authorities describing their course differently. To take an instance: Swedenborg says, "The superior petrosal sinuses *seem* to admit, in addition, the fluid of the *second* order, which passes through the bones of the skull." Now the only channel through which the superior petrosal sinuses can receive such blood is through the anterior occipital or transverse sinus, which opens into the cavernous sinuses in close proximity to the mouths of the above sinuses. According to Virchow, Sapolini, Vicq d'Azyr, and Sappey, this arrangement is found, and the blood, therefore, may pass that way. But on the other hand, such a communication between the anterior occipital or transverse sinus and the superior petrosal is totally ignored by Bell, Henle, Knott, and Langer; while Quain's drawing (eighth edition) makes the transverse sinus unite, not the superior petrosal, but the inferior petrosal sinuses! One very likely explanation, no doubt, of these differences of description may be that the arrangement of these sinuses varies in different cases, and anatomists are not surprised at finding a considerably greater variation in the course of arteries and veins than, for example, of nerves. Swedenborg's theory is, however, so complete that it is necessary to pay attention to every point in the anatomy of the cerebral parts and of the sinuses, very important considerations arising, as has been seen, out of small variations in arrangement. Obviously, if the pituitary gland's function is an enigma or a myth, it makes not the slightest difference in our minds how the sinuses near it proceed; but it is quite different when a detailed theory is held regarding it. Dr. Tafel's opinion, at the conclusion of his elaborate 'Note on the Pituitary Gland: its Sinuses, etc.,' is that Swedenborg's "theory has passed triumphantly through all the crucial tests to which it has been submitted." Our own opinion, after time has been allowed for recovery from the shock of supposing it possible that a metaphysician, an ontologist, or theologian, could by sheer insight and force of inductive reasoning teach anything whatever to anatomists and physiologists regarding things in their own department, undoubtedly is that Swedenborg and his editor have proved completely that the pituitary gland is a gland engaged in the elaboration of the blood, whatever else it may be; and that in the other things which he says about it, even

if he has not proved them, he has put a strong case for respectful consideration.

Besides the function of the pituitary gland, this volume deals, as another of its main subjects, with the cerebro-spinal liquid; and in Note VII. the Editor sums up the evidence for Swedenborg's views regarding it. It is generally supposed that Cotugno, in 1764, first demonstrated the existence of this fluid as filling up the large interstice between the dura mater and the spinal cord from the occiput to the os sacrum, and also all the interstices between the brain and the dura. But Swedenborg, writing in 1743, was well acquainted with this fluid. Thus we find him saying, "The arachnoid membrane may be separated from the subjacent pia mater by injection and a blow of air, and in dropsical brains it actually appears separated; whence it follows that a humour flows between it and the pia mater, by which they are kept apart and prevented from coalescing." His idea was that a subtle liquid was derived by the alternate expansion and contraction of the nervous matter of the cerebrum, cerebellum, and cord (synchronous, as he showed, and as has been demonstrated since by Mosso, not with the cardiac, but with the respiratory motion) into and between the fibres of the brain, and the fibres and fascicles of the nerves proceeding from the brain (as e.g. the optic nerves), and the fibres and fascicles of the spinal nerves. This fluid is supposed to bathe the fibres, so as to keep them moist and to enable them to perform their functions in a proper manner. The cerebral fluid he supposed passed from the lateral into the third ventricle, and thence, as we have seen, into the pituitary gland and the infundibulum, to find its way into the cerebral sinuses and veins. The great mass of the cerebro-spinal fluid however came, he believed, from the cerebellum, and found its way by the occipital opening into the fourth ventricle. (It would, it seems, therefore be better to name it the cerebello-spinal fluid than the cerebro-spinal.) From the fourth ventricle it passed into the sub-arachnoid space. Thus he says: "The lymph of the fourth ventricle and of the choroid plexus is discharged into the duplicature between the pia mater and the arachnoid membrane; and thence through the continuous and customary ducts and follicles of the arachnoid membrane. It is derived especially into the posterior part of the medulla oblongata, where that membrane floats about quite densely and loosely." The history of the discovery of the cerebro-spinal fluid is very curious, even more so because so much more important than that of the canal of Tabarrani formerly referred to. Swedenborg knew of it in 1743. "Haller came very near discovering it" (say Key and Retzius, who, however, do not appear to have studied Swedenborg, their own countryman); "but to Cotugno is due the honour of having discovered this fluid as a constant formation occurring in the living subject under entirely normal conditions. Twenty-two years later, Sommering knew no more about it than what is contained in his expression, that "between the arachnoidea and the pia mater there is often discovered a phlegmy water." Magendie

it was, however, who re-discovered the fluid, and showed that it is contained in the subarachnoid spaces on the surface of the brain and cord, as also that it is in a constant state of motion with the respiration (1825). He also thought it was an exudation from the vessels of the pia mater. Luschka however (1858), with whom Key and Retzius appear to agree, thought that the choroid plexuses of the brain are the genuine secretory organs of the fluid. Swedenborg's view was that the liquid was secreted by the blood-vessels of the pia mater, by the pia mater itself, and by the choroid plexuses; and we think it cannot be denied that these opinions, expressed 21 years before Cotugno, 82 years before Magendie, and 142 years before Luschka, Key and Retzius, show a remarkable grasp of the subject, and a profound philosophic instinct and power of inductive reasoning.

According to Swedenborg, also, the valve of Vieussens has for its general function the prevention of the mixing of the cerebral with the cerebellar fluid. That the two fluids must be quite different appears from several facts; but the following one is very striking, viz. that the fluid of the lateral ventricles of the brain is characterised by containing salts of potash, while the fluid of the fourth ventricle contains preponderatingly salts of soda (Hoppe, 1854). Swedenborg thought that in some circumstances the cerebro-spinal fluid flowed back and mixed with the fluid coming from the lateral ventricles. He is probably correct in this opinion, and in thinking that the valve of Vieussens does not absolutely and in all circumstances shut the two fluids off the one from the other; but it seems to us that some of the reasoning by which he supports his view is of very unconvincing character. He says that the cerebro-spinal or cerebellar fluid may sometimes mix with the cerebral or ventricular fluid (for we suppose "cerebellar" on page 553 is a misprint for "cerebral"), because these two fluids are both contained in the red blood, and being derived from the same source they may, he says, be of assistance to each other, and thus be commingled. Even Dr. Tafel's acute intellect seems to have been misled here, for he appears to accept the reasoning. Yet what should we think of a physiologist who should argue that because bile and urine are both contained in the first place in the red blood, they may therefore, because "derived from the same original source," "be of assistance to each other, and thus be commingled"!

Swedenborg held that the cerebellar or cerebro-spinal fluid passed from the fourth ventricle (which, he said, had no duct) by a duplicature between the arachnoid and pia mater, forming a kind of cleft at the end of the calamus scriptorius. The existence of this foramen may now be said to be admitted by scientific men, mainly owing to the demonstrations of Key and Retzius, who proved its existence by injections which they made from the subarachnoid spaces as well as from the ventricles. They used congealing injections, which furnished complete casts of the foramen and surrounding spaces. The history of this foramen is very interesting, and is given by Dr. Tafel. Haller asserted its exist-

ence, Monro (1783) denying that it was a real foramen, while Magendie re-directed attention to it, and his views were accepted by Ecker and Cruveilhier (1843), by Luschka (1858), and Stilling (1870). But Krause (1843) and Reichert (1861) said it was an artificial and not a real opening; while Kölliker's statement to the same effect counted for a great deal. Now, however, Quain, in the 8th and 9th editions of the 'Anatomy,' declares for its actual existence. M. Sée, so lately as 1879, contends that it is of very small importance, Dr. Tafel attempting, however, and apparently with effect, to answer his objections. We may observe while on this point, that Key and Retzius, on whom Dr. Tafel largely counts for the maintenance of Swedenborg's views regarding the course taken by the cerebro-spinal fluid, speak of the fluid *entering and leaving* the fourth ventricle. The statement certainly is not made of their own views, but of those of Magendie, but it is made seemingly without demur. We confess to having been greatly confused by this statement, for if Swedenborg's views are correct, the fluid may *leave* the ventricle indeed, but can hardly be expected to *enter* it, since it flows from cerebellum to nerves, and not from nerves to cerebellum. This point is discussed by Quincke, who experimented on living dogs, and found that it was impossible for a current of injection to find its way in the living animal from the subarachnoid space, though it was comparatively easy to send it from the fourth ventricle into the subarachnoid space. The remark, however, made by Key and Retzius (in itself an unimportant one) is of consequence, as showing the want of definite opinions even among those anatomists on whom Dr. Tafel rests the most strongly for the proof of his positions, and whose opinions indeed furnish, it will be at once conceded, the most conclusive evidence for or against the side they take.

Besides the foramen of Magendie near the bottom of the ventricle, Luschka discovered in 1855 two openings at the angles of the fourth ventricle, leading from it into the subarachnoid spaces; and Key and Quain agree with him; while M. Sée, though admitting the existence of the openings, does as in the case of the foramen of Magendie and minimises their importance—another proof of what we have just said, that physiologists have been by no means certain as to the uses (or even the existence) of these structures.

These foramina, it may be admitted, are very small; so that there might be differences of opinion as to their uses, and even as to their presence. But whoever wishes to see how far differences as to the existence of things (differences, that is, as to actual existences, depending in turn on the testimony of the senses) can be honestly held, ought to familiarise himself with the views that have been held by different anatomists as to the existence of a central canal in the spinal cord. According to the views of Swedenborg there ought to be such a canal, for he thought that a very refined kind of liquid circulates in the spaces by which the individual fibres in the medulla of the cord and by which the nerve-roots depart. This fluid was, in his view, finer and more subtle than that which

passed through the foramen of Magendie and through the foramina of Luschka (though of course it is an anachronism to make Swedenborg speak either of Magendie or Luschka), and it required therefore a special channel for its passage; and in reference to this Swedenborg says, "Whether such a channel open immediately from the calamus scriptorius into the medullary portion of the spinal cord, to my knowledge has not yet been discovered. For this purpose the fourth ventricle is contracted into the narrow form of a goose-quill." A canal of this kind is required for ministering properly to the uses of the cerebro-spinal liquid, and also to enable the spinal cord to go through its general systaltic and diastaltic motion in connection with the cerebrum and cerebellum. The more recent views on the subject of this canal are curious, and although there is not space for a full statement of them, it may be said generally that many authorities have asserted its existence and many have denied it. In Swedenborg's time Morgagni had said that some human bodies had the canal, and quoted the case of a Venetian fisherman which showed it. Gall and Spurzheim in the beginning of this century, and more recently Stilling and Wallach, asserted its presence. Solly denied it, and so did Dr. Todd. Still it is now accepted as a fact, and, for instance, Quain in the eighth edition describes it as "a minute central canal, which, in prepared sections of the cord, is barely visible, as a speck, with the naked eye." It is surely remarkable that Swedenborg's views should have led him to expect that a cavity, not yet known to exist, would in the future be discovered; and that his anticipation should be verified.

As to the future course of the cerebro-spinal fluid, Swedenborg's view was that its function was to moisten and lubricate the spaces which exist between the fibres and the fasciculi in the interiors of the nerves. He held generally, that the fibres themselves were pervious; that between the fibres interstices were found; that other interstices, but longer, existed between the fasciculi of the fibres, and also between the general coatings of the nerves themselves. Now modern science entirely bears out the existence of spaces in the cranial and spinal nerves which are continuous with the subarachnoid space of the brain (or cerebellum) and cord. This may be stated on the authority of Cotugno, of Key and Retzius, and of Bogros (1827), who by injections of mercury found that the nerves are pierced by a comparatively narrow canal open to injections, and bounded by the various neural membranes. Cruveilhier (1845) repeated these experiments and confirmed them, and they have been amplified by Key and Retzius. Still it was a long time before Cotugno's view, though correct, found its way into modern science; and on this point Dr. Tafel offers the interesting explanation, that it was Bichat who retarded progress by stating that the arachnoid had the same general arrangement as the serous membranes, and that, as it consisted of a parietal and visceral layer, it must necessarily hinder, at its duplicatures, the passage of fluid from the arachnoid cavity along the nerves. This view obtained up to ten years ago; but the text-books now

describe the real arrangement. The contrast between the statements contained in the eighth and ninth editions of Quain's is striking, as the former details the closed-sac arrangement of the arachnoid, and the latter the real one.

The interesting chapter on the laws of motion of the cerebrospinal fluid shows that the fluid rises during expiration, and falls during inspiration, and not *vice versâ*, as some have supposed. As to the fluid itself, Swedenborg held that it consisted of three sorts or "orders," as he terms them. The first order, or spirituous lymph, courses in the interstices around the individual fibres, for the fibres are pervious; and, by the way, he seems to think that each fibre is continuous from its origin to its termination. If this were so, plainly the cord would require to be built up like a cone, since all the fibres that go to all parts of the body would have to pass from the upper part of the cord, a less number from the middle part, and the least number from the lowest part. But the cord has not this structure, swelling out at some parts and diminishing at others; with more relation to the number of nerves that have origin at those parts than to position in the cord. The fluid of the second order, the subarachnoid or cerebro-spinal liquid proper, flows around the nerve-roots or fascicles, which are composed of the individual nerve-fibres; while the fluid of the third order, the subdural fluid, passes through the spaces in the general coating of the nerves. Through these latter spaces, according to Swedenborg, there circulates also the fluid of the second order. As to the fluid of the first order, the existence of this has been confirmed by Obersteiner (1870) and Bevan Lewis (1877). The last made out that the nerve-cell is bathed in a constantly renewed current of lymph on all sides, and confirmed the position of Swedenborg that "a subtle liquid is distilled from the least arteries of the brain," which liquid "flows outside the cortical glandules." Dr. Tafel enters minutely in his note into the structural mechanism by which this fluid is conveyed, drawing as usual, upon modern authors for his account of it, and the reader will find his remarks of great interest. The fluid of the *second* order is partly cerebral, partly cerebellar. The former is carried partly into the nerves through the cribriform plate of the ethmoid bone, and partly is forced into the Pacchionian bodies, which are really *villi of the arachnoid*, and thence finds its way into the sinuses. The cerebellar portion of the fluid of the second order finds its way into the fourth ventricle, being prevented from passing back into the third ventricle by the valve of Vieussens, and is urged into the subarachnoid space about the medulla, and thence into the subarachnoid spaces of the departing cranial nerves. The subdural juice or fluid of the *third* order is also partly cerebral and partly cerebellar. Some of the cerebral portion finds its way into the mucous membrane of the nose, and some also into the Pacchionian bodies. In this way the subarachnoid or fluid of the *second* order mingles, to some extent, with the subdural or fluid of the *third* order. Lastly the cerebellar portion of the fluid descends into the subdural space of the cord,

and is conveyed finally into the subdural spaces of the peripheral nerves. A section on the universal circulation of the cerebro-spinal fluid concludes Dr. Tafel's elaborate note, and will be found full of information.

We have left no space to deal with the pathological aspects of this most important subject. There is the less reason to do so, that Dr. Tafel promises to take it up later, when we hope our review may have the opportunity of following him. Mainly we believe the subject is at present a blank, at least to the vast majority of medical men. Who, for instance, ever heard of a diagnosis being made of inflammation of the pituitary gland? Or who ever suggested that anæmia may sometimes be due to disturbance of its blood-making function? Yet who shall say that these conditions do not frequently occur? Ideas have ever taken time to assimilate, and evidently it will be necessary that the state of general opinion must undergo considerable alteration before we are in a condition to diagnose or treat such conditions. An important step in this direction will be taken when the profession are familiar with this work of Dr. Tafel; and it appears to us that the result to the profession will be as important if they accept his views, as if (what does not appear possible) his opinions should be in all respects confuted.

A. Rabagliati, M.D.

Studien über Aetiologie und Pathogenese der Spontanen Hirnblutungen. Von Dr. Leopold Löwenfeld. Wiesbaden: Bergmann, 1886, 1 vol. 8vo, 166 pp.

Dr. Löwenfeld has contributed an important and exhaustive treatise to the literature of Cerebral Hæmorrhage, which deserves a prominent place in the same. The large part which original work plays throughout must render the volume one of practical value, and his thorough research into the subject in every aspect is amply vouchsafed for by the extensive Bibliography which is apparent.

Dr. Löwenfeld has not been willing to pander to his readers, by sacrificing a complete elucidation and discussion of the almost innumerable theories which have always attended Apoplexy, to brevity; and here in a little want of conciseness he is perhaps at fault. But since undoubtedly his work must rank as a text-book on Cerebral Hæmorrhage, this discardance of brevity is pardonable, and those who may make the latter subject their field of research will be grateful to the author for having so carefully prepared the ground for them.

In part with the volume, are the very excellent plates which are appended thereto.

It is divided into eleven sections. We propose noticing these in succession as briefly as the contained matter will allow.

The first two deal respectively with the Literature and Etiology of Cerebral Hæmorrhage from the earliest times to the present. The matter, interesting as concerning the progress of theories, does not here concern us.

The third section first claims our attention. It deals with the examination of the intracerebral vascular system.

With the view of thoroughly acquainting himself with the different forms of vascular changes, the author examined a large number of the brains of individuals dying from different causes, as also those of animals. Of brains the seat of spontaneous hæmorrhage he examined seventeen, many hundred vessels were carefully observed, and the condition of those taken from the walls of apoplectic foci especially investigated.

The method employed in the preparation of sections and specimens is fully described; the fresh preparation being especially insisted upon, as by far the preferable. His descriptions, except where the contrary is stated, are made from these.

There follows a concise and lucid account of the normal structure of the arteries, a few points in which demand special mention. Regarding the structure of the adventitia, the author expresses himself thus: "The various formations met with on the outer side of the muscular coat are only forms of the adventitia which occur by folds in the same. I must emphatically assert that I never could convince myself, by most careful examination with high powers, that connective-tissue fasciculi are interposed between the adventitial sheath and the muscular coat. That which offers the appearance of such an intermediate layer, and was mentioned by others as such, is merely very fine, closely-pressed folds of the adventitial coat." He also alludes to the extreme variation in the amount of nuclei found in the adventitial sheath, and of the contents of the lymph space, in healthy vessels. He disputes Obersteiner's and others' statements regarding the presence of a perivascular space, also of the existence of prolongations from the adventitia into connective-tissue cells in the brain cortex.

Of morbid conditions of the vessel coats, the following alone appear to call for note.

The author believes the cell-accumulation at times present in the external coat to be dependent upon an obstruction to the lymph-flow and resulting coagulation; this view being favoured by the occurrence of these cell-collections at the bends and tortuosities of vessels where the lymph-current might the more easily be stayed.

According to him, the blood in dissecting aneurisms occupies the adventitial space (as was held by Laennec). His investigations into the origin of the blood extravasation differ in their result from that generally accepted. From his sections he was able to satisfy himself that no breach in the muscular coat or intima existed. In successive sections of an aneurism he was able to trace the fading, even disappearance of the muscularis, and the extreme thinning of the fenestrated membrane, but not to discover any rupture. In adjacent parts the muscularis showed red and

white blood corpuscles in its midst, as if "surprised" in the act of emigration. He inclines to think that osmosis may account for the blood effusion without a rupture in the intima. However, he concludes that perhaps a very small breach of continuity may occur, through which blood may exude; coagulation sealing up the opening. A thickening of the adventitia which might account for its not tearing under the pressure of blood, he has not observed.

Of granular degeneration of the muscularis the author speaks at some length, on the grounds that it is a change but little recognised, Obersteiner alone having made mention of it. It has been found in a large number of apoplectic brains, not infrequently in the aged, and he inclines to count it amongst senile changes. It however exists not seldom in the brain-arteries of heavy drinkers, non-apoplectics.

The degenerative process is quite distinct from any fatty change, no trace of the latter being discoverable. Regarded by Obersteiner as a pseudo-hypertrophy of the muscle layer, from the marked increase in bulk conjoined with a degenerative process, it is considered by Löwenfeld to indicate a true granular change: he is careful to draw distinction between it and the hypertrophy affecting the muscularis of the small vessels (described by Johnson Ewald). The intima usually undergoes a corresponding degeneration in the diffuse form of the disease, whilst in the circumscribed variety, atheroma at times accompanies.

In the portion of the third section devoted to Miliary and Diffuse Aneurisms, he classifies dilatations of the vessels in six forms—

1. Those occurring at the division or branching of arteries.

2. Obersteiner's "Rosencranzform," or chaplet form, of presumed vaso-motor origin; found by the author fairly often in the vessels of apoplectic brains.

3. Those due to spasm of groups of muscular fibres, an exactly opposite condition to the preceding, and probably dependent on a P. M. change.

4 & 5. Scattered circumscribed swellings, with and without structural change in the walls.

6. Diffuse ectasie over some extent of vessel. Only 4 and 5 can be considered as miliary aneurism. In respect to the size of the latter, the author has met with them from purely microscopic dimensions to those of a lentil, and considers that a hard and fast line cannot be drawn between these aneurisms of the basal arteries and of the meningeal vessels. As might be assumed from this, he has found them present in arteries of every size down to the branches of anastomosis. The smaller preponderate, and extremes of size are seldom found. The most frequent site at which they exist is the neighbourhood of an apoplectic focus—therefore in the basal ganglia. He revises the order of the localities in which they occur to the following: optic thalami, corpora striata, centrum ovale, convolutions, pons and cerebellum. Diffuse ectasie in apoplectic brains have not attracted much attention. The expansion of all three coats of the artery extends sometimes to

over 1 c.m. in length, such expansions occurring over large tracts of vessels. Found in the vicinity of miliary aneurisms, it is not always easy to distinguish them from the fusiform variety of the latter.

Regarding the theories advanced as to the origin of miliary aneurisms, the author opines that the views held by Zenker and Eichler, attributing their causation to atheroma of the intima, are not tenable, and for the following reasons:—

1. Miliary aneurisms are found which do not show a trace of atheroma of intima.

2. Advanced atheroma occurs without any swelling of the vessels; of this the author has assured himself in hundreds of preparations.

Nor does he believe that the muscular coat exclusively can cause them, because although the muscular tunic is the chief agent in preserving the calibre of the artery, and degenerative changes may therefore be expected to result in expansion of the vessel, still this is not always the rule in miliary and allied aneurisms; on the contrary, very often complete destruction of the muscularis is found in vessels showing no dilatation, a normal state of the adventitia being coexistent. He considers two conditions may be adduced as causes of miliary aneurism, both of which are probably operative.

(*a*) A local increase of blood-pressure.

(*b*) A local change in the vessel wall resulting in diminished resistive power.

Conditions which can lead to a local increase of blood-pressure are by no means frequent in miliary aneurisms. If, however, the emergent artery of the aneurism becomes blocked, as at times it may, from the dislodgement of products of disintegration, in terminal arteries as those of ganglionic area, a local increase of blood-pressure must result, and may account for a further expansion of the aneurism, originated by degenerative change. When we consider that the vessel coats in the vicinity of the aneurism must be in a condition to withstand the blood-pressure, or at least to offer a greater resistance to it than those of the dilated portion, and since the disease of the vessel walls is never limited to the region of the miliary aneurism, but is diffused around, so the latter indicates the site of an earlier change, but there is no reason to suppose that the muscularis was this site. It follows from this that alterations of the muscularis can only occasion miliary aneurism when limited to a circumscribed spot, or when they advance more quickly than in surrounding parts.

Such instances of miliary aneurisms as occur without obvious wall-changes leave their origin to two assumptions—

(1) The changes are such as cannot be recognised with our present modes of research.

(2) They arise from vaso-motor disturbance, paralysis of the vaso-motor apparatus or stimulation of the vaso-dilator. Both in miliary and diffuse aneurisms the possibility is allowable, that

the dilatation is caused by a vaso-motor lesion, the structural changes having occurred secondarily.

The condition of the veins is analogous to that of the arteries. In the capillaries, hyaline and fatty changes were the most often found. In arteries undergoing fatty or granular degeneration, a hyaline transformation in the vasa-vasorum was frequently observed. Its influence on the state of the arteries is probably not more than that of furthering existing changes. The consideration of the relationship of the different vessel alterations concludes the section. As regards the muscularis, fatty degeneration was the most constant change; atrophy and granular disease following in succession of frequency. The intimal changes were usually commensurate with those of the media; fatty degeneration and atheroma were never quite absent. In the adventitia, nuclear prolification and thickening were over some part of the vessels always present, though seldom markedly. Miliary aneurisms were never entirely absent, their number varying from a few to some hundreds; the diffuse ectasie were sparsely present, their usual site of occurrence was the wall or environ of an hæmorrhagic focus; here also dissecting aneurisms chiefly located themselves. Certainly the author's observations do not corroborate Charcot and Bouchard's views with regard to miliary aneurism, neither as to the part they play in the causation of the hæmorrhage, nor to the mode of their formation, since the latter observers consider their origin due to a form of periarteritis, whilst Dr. Löwenfeld has found that only in a moderate number of these aneurisms are the adventitial changes present; whilst again, though not frequently, they were found with perfectly intact coats. In Section IV. their causal influence as regards hæmorrhage is alluded to. Of the periarteritis, which Charcot and Bouchard claimed to have observed over the intracerebral vascular system, the author is not aware; neither himself nor other German authors having ever seen it. Nor can he explain the complete atrophy of the muscularis which they describe. Where such occurs, it is not secondary to adventitial changes, since its alterations do not run parallel to those of the latter.

Neither can he endorse Turner's views as regarding inflammatory softening of the vessel wall, since the cell accumulation which Turner accepts as a sign of an inflammatory process may be only due to a mechanical cause, such as lymph-engorgement, or to the changes undergone by vessels after the occurrence of a proximal hæmorrhage.

In the Fourth Section are considered the points of exit of the bleeding. The author does not agree with Charcot and Bouchard concerning the importance of miliary aneurisms in the genesis of hæmorrhage, but rather believes that the unexpanded vessels are the source of the same, and though Charcot was probably not wrong in arguing that in the walls of fresh blood foci, burst miliary aneurisms can always be found, still in the majority of these cases they were present in other parts of the brain, so that their occurrence in this site had no special significance.

The veins may also give rise to small extravasations. Concerning the relations of atheroma of the basal vessels and great arterial trunks to cerebral hæmorrhage treated of in the Fifth Section, there is but little to note. The basilar most often presents disease, only escaping in one-seventh of the cases above æt. 60. The intracerebral vessels and basal are affected inversely as regards extent. There is but an occasional relation between atheroma of the basilar and cerebral hæmorrhage, and what applies to the basilar does so equally to the aorta, etc.

In the Sixth Section is discussed the relation between kidney disease and cerebral hæmorrhage. Important differences are exhibited in kidney disease with and without cerebral hæmorrhage. In the latter, changes in the adventitia and intima take the lead, those in the muscularis being subsidiary. But in cases of the former, the muscularis is the most involved, as shown by its extent as an independent condition, thus evidently not being a mere aggravation of the change belonging to the kidney disease but a development in another direction. The author discusses at length the value of cardiac hypertrophy, blood alterations, toxæmia, heredity, and lastly chronic interstitial nephritis in connection with the accompanying greatly-increased penetrability of the vascular system. Of much influence, as the latter disease is, on the occurrence of hæmorrhage, another factor is required, since hæmorrhage does not happen in all cases of interstitial nephritis. An *à priori* condition of the vessel walls is assumed therefore. (Heredity, hæmorrhagic diathesis.) Blood stasis is also of importance in the localisation of the extravasation. Thus in chronic nephritis are two forms of cerebral hæmorrhage met with—

1. Moderate hæmorrhage in few or scattered foci, explained by ruptured vessels.
2. Small multiple extravasations, without arterial rupture; the expression of a hæmorrhagic diathesis.

Regarding the influence of cardiac hypertrophy and disease, there can be no doubt that, arterial disease being present, the increased blood-pressure must be an important factor in determining a speedier rupture. Such increase of pressure is occasioned, though in a different manner, by failure of the heart's propulsive force, so that degeneration of the muscular texture and valve disease, not compensated for, are important features likewise. Embolism only needs mention. In nearly half the cases of apoplexy, total or left-sided cardiac hypertrophy was found.

Section VIII.—Concerning the relations of alcohol, lead, gout, rheumatism, and syphilis. The toxæmia of lead is considered to have as direct an influence as that of alcohol, and it seems not improbable, according to the author, that cerebral hæmorrhage may be due to such lead toxæmia without concomitant renal atrophy or cardiac hypertrophy.

In support of his views he adduces Mayers' experiments on animals, and a case observed by him, in which lead toxæmia without renal disease seemed to be pointed to as the cause of the

fatal hæmorrhage which occurred. Neither gout nor syphilis does he regard as playing important parts in the ætiology of cerebral hæmorrhage, seldom indeed can they constitute a cause without auxiliary conditions. Rheumatism, by reason of the endocardial and valvular complications, holds a more prominent place.

The ninth and tenth sections concern respectively the Habitus Apoplecticus, and the influence of the nervous system upon hæmorrhage. In the former, there is nothing requiring mention; that the so-called apoplectic build no longer bears the same significance that once it did, is well recognised, as is the relationship of malnutrition. Corpulence, except when combined with senile vascular changes, appears to have likewise no influence. No great import as yet can be attached to the part played by nervous disturbances.

Section XI.—The majority of this is occupied with the consideration of the transmission from the parents to the offspring of certain conditions of the cerebral vessels, independently of a general circulatory participation, which predispose to hæmorrhage. The theory of greatest probability is that in such conditions exists a defective development of the brain vessels, since the development of neither the latter nor of the brain proceeds parallel with that of the general circulatory system. A normal constitution of the cerebral vascular system can only be said to be present when it bears a certain proportion to the brain weight. So that where this proportion falls below a certain standard, defective development may be said to exist.

With a view to determine the relations between the cerebral and general vascular systems, and the weight of the brain, the author measured in 122 cases (in which, so far as could be ascertained even microscopically, no disease was present) sections of the basilar, carotid, and vertebral arteries, in terms of the hundredth of a centimetre. These were always made at the same level and the effects of post-mortem rigor eliminated. The results were suggestive, but could not be embodied here. The proportion of the cerebral arteries to the brain weight and systemic vessels was very variable, and a defective development of the first could exist with a normal development of the last. We may accept such a local deficiency as a possible powerful factor in predisposing to disease, nor can we doubt that in the majority of cases such factor operates. For, with a given blood-pressure, narrow vessels must experience an increased distension, and thus suffer injury before the well-developed ones.

What variations in the thickness of the coats, independently of the vessel circumference, occur under normal conditions, investigations have not as yet convinced us of. An increase is found as a senile change, and with general disturbance of nutrition. That the muscular coat, as has been said, is the seat of arrested development, we have no justification in assuming.

In conclusion we must allow, that the alterations in the cerebral vessels of apoplectics, in by far the majority of cases, are brought about by factors which emanate from the blood. Such factors are

probably, in great part, of a mechanical nature, and operate in the form of a powerful dilatation and rending of the vessel walls by the blood impulse; in part also are founded upon alterations of blood.

Mechanical operations can be induced by very different events in the circulatory system—

1. Through a more forcible action of the "Pumpwerkes" (absolute increase), cardiac hypertrophy (relative increase), marasmus with participation of the vessel-walls without corresponding involution of the heart

2. Through obstruction, (*a*) arterial system—sclerosis, atrophy of kidneys, obesity, habitual constipation, sedentary life; (*b*) vascular system—myo-degeneration of the heart, uncompensated failure of valves, and emphysema.

3. Through increase of the contents of the vascular system, e.g. plethora.

The blood alterations which induce disease of cerebral vessels are—

1. Chemical: marasmus, nephritis, obesity, gout? alcohol, and lead intoxication.

2. Parasitic: Syphilis, rheumatism?

F. St. John Bullen.

Die Morphiumsucht und ihre Behandlung. By Dr. Albrecht Erlenmeyer.—Third edition, 1887.

The importance of the subject Morphinomania and its treatment is shown by the number and size of the works now written upon it, as well as by the earnest study which some of them give proof of. Dr. Erlenmeyer has given a lucid exposition of the origin, symptoms, treatment and prevention of the disease, and the reader will feel as he peruses the book that the author is speaking from wide experience.

He commences in his first chapter by a consideration of the causes which engender the morphia habit, and shows that only certain individuals, on whom the drug works in a particular fashion, are liable to become *habitués*. In some persons even small doses produce such unpleasant effects that they have no desire to repeat the experiment. Those, on the contrary, who are liable to become victims of the habit, experience very pleasant sensations, which the author terms "Euphorische Wirkung des Morphium."

In the second chapter he considers the pathological anatomy of the disease, which, as might be expected, has nothing which is characteristic, except the local evidences of subcutaneous injection, when the drug is taken in that way.

The third chapter contains an enumeration and review of the

long array of symptoms referable to body and mind which morphia *habitués* present. The author records some curious facts showing that children born of mothers who are addicted to the vice are affected in just the same way as if the drug had been directly administered to them: they are in fact morphia *habitués*, and are liable to serious troubles as soon as they are separated by birth from the source of their constant supply.

With regard to the intermittent fever to which Levinstein drew attention, Dr. Erlenmeyer believes that it has nothing to do directly with the morphia, but that it is due to the abscesses and other inflammatory conditions caused by the injecting needle.

Nor does he consider that albuminuria results from the habit, nor yet from the discontinuance of it, except when the latter is suddenly effected, and produces delirium; the albuminuria he then attributes to the delirium, and not to the withdrawal of morphia.

In describing the symptoms produced by suddenly stopping morphia, he draws particular attention to collapse, which indeed is the only grave objection to this method of treatment. It is a very serious matter, and can only be remedied by an injection of morphia.

In the 'Lancet' for June 25th, 1887, Dr. Oscar Jennings of Paris wrote a paper on "The Relief of the Morphia Craving by Sparteine and Nitro-glycerine," and the praise with which he speaks of the effects of these drugs in the treatment of patients of this class must raise the hopes of medical men. For although a certain amount of relief may be afforded by one drug or another to those who are being broken of this habit, every one must allow that to produce anything like comfort during that trying period has been beyond the power of medicine. Oscar Jennings bases his treatment on sphygmographic indications. He describes an excessive vascular pressure, producing a "plateau" in the pulse-tracing, as occurring with the withdrawal of the drug: to anæmia, as represented by this contraction of vessels, he attributes the craving with its attendant horrors, and he gives sparteine and nitro-glycerine, which by removing the "plateau" produce complete relief. It was therefore with some curiosity that we turned to Dr. Erlenmeyer's observations on the pulse. It is true that he appears not to have used sparteine and nitro-glycerine, but he goes with great care into the condition of the pulse, and gives many sphygmographic tracings. The results he arrives at are precisely the reverse of those which Oscar Jennings records. He says, in fact, that during the period of abstinence the pulse-tracing shows paralysis of the vascular wall and diminished pressure; and that if an injection be then given, contraction of artery with increased pressure result. He then adds, that the pulse in those suffering from the morphia craving is not characteristic; sometimes its peculiarities are those described, sometimes they are not.

In Chapter IV. the diagnosis of the disease is fully considered, and the examination of the urine for morphia, which used to be relied upon, is now said to be quite unreliable. In the fifth chapter he

discusses the question of treatment. He discards the sudden system practised by Levinstein, because of the liability to sudden collapse which attends it. This, however, he allows is the only disadvantage of the method in question; still, if this complication be anything but a most unusual one, it must prove a fatal objection to this form of treatment.

The gradual method Dr. Erlenmeyer considers uncertain on account of the constant supervision necessary to ensure abstinence from the drug, and he considers it further undesirable because of the prolonged period of misery which it entails. Best of all, he says, is the rapid withdrawal of the whole quantity of morphia in from six to twelve days. By this means are ensured certain success, absolute freedom from danger, and a short period of suffering.

The author devotes many pages to the question of the treatment by cocain. His conclusions are—1. That its use does stop the craving. 2. That this effect is very transitory, only lasting from 10 to 25 minutes. 3. Cocain is a real substitute for morphia and not an antidote. 4. It should not be given, as its effect is so temporary and its use dangerous; moreover, there is great risk of developing a habit, "Cocainismus," which is even worse than morphinomania.

Chapters VI. and VII. contain considerations with reference to prognosis, and to certain legal questions. Chapter VIII. gives an account of fifty cases, and Chapter IX. a carefully compiled literary history of the disease.

Dr. Erlenmeyer has handled the whole subject clearly, scientifically, and practically, and has produced a work which can be warmly recommended to all who are desirous of knowing about the disease, or who require guidance in the treatment of its victims.

SEYMOUR J. SHARKEY.

Abstracts of British and Foreign Journals.

Current Nerve Anatomy and Physiology.—Titles and Indication of Contents of Papers which have appeared during the Quarter (Midsummer to Michaelmas, 1887). By ALEX. HILL, M.D., Downing College, Cambridge.

HUMAN BRAIN.

1. Observations on the Brain of Man. MICHEL VON LENHOSSÉK (*Anat. Anzeiger*, 1887, No. 14, June 15).

Three papers. 1. Description of a delicate white bundle on the outer side of each corpus albicans, already mentioned but not described by Von Gudden, for which the author proposes the name Stria alba tuberis. It is derived from the descending pillar of the fornix. 2. A description of the structure of the corpora albicantia as studied in section by Weigert's method. Distinction of nuclei. 3. A new bundle on surface of the pons; Fasciculus rectus pontis.

2. The Brains of Criminals (Parieto-temporo-occipital surface). Second memoir. LOR. TENCHINI (*Luigi Battei, Parma*, 1887, 14 *plates*).

COMPARATIVE ANATOMY.

3. Researches into the Structure of the Brain of Mysis Flexuosa, *Müll.* M. KOEHLER (*Ann. des Sciences Naturelles, Zool.* t. ii. pts. 3 and 4).

Examined by means of sections in various planes.

4. The Nervous System of the Opheliaciae. DR. WILLY KÜKENTHAL (*Jenaische Zeitschrift*, vol. xx. hft. 4, p. 511).

5. Comparative Anatomy of the Nervous System of the Isopoda. E. BRANDT (*Horæ Soc. Entom. Russicæ*, t. xx. p. 245. *Russian*).

6. The Dipnoan Brain. BURT G. WILDER (*American Naturalist*, 1887, p. 544, *June*).

MORPHOLOGY.

7. The Ontogeny and Phylogeny of the Torus Longitudinalis of the Mid-brain of Bony Fishes. RABL-RÜCKHARD (*Anat. Anzeiger*, 1887, No. 17, p. 549, *August* 1).

The structure belonging to the mid-brain of bony fishes, and known as the Torus longitudinalis, is not homologous with the fornix or other constituent of higher brains, but is present in the brains of most vertebrates in a rudimentary form as an ependyma development.

8. The Homologies of the Chorda Tympani in Lower Animals. FRORIEP (*Anat. Anzeiger*, 1887. No. 15, p. 486, *July* 1).

9. On the fate of the Muscle Plate and the Development of the Spinal Nerves and Limb plexus, in Birds and Mammals. DR. A. M. PATERSON (*Quart. Journ. Micro. Sci.*, vol. xxviii. pt. 1, p. 109, *Aug.* 1887, pl. vii. and viii.).

10. Note on the Ciliated Pit of Ascidians and its relation to the Nerve Ganglion and so-called Hypophysial Gland, and an account of the Anatomy of Cynthia rustica (?). LILIAN SHELDON (*Quart. Journ. Micro. Sci.*, vol. xxviii. pt. 1, p. 131, *Aug.* 1887, pl. ix. and x.).

The function of the ciliated pit is considered to be aëration of the brain, and, secondarily, it serves as a duct for the so-called hypophysial gland. The ciliated pit itself, however, is regarded as probably homologous with the hypophysis of vertebrates, which, according to this view, is an atrophied organ the function of which is lost.

11. Morphological Value of the Lateral Nerve of Petromyzon. CH. JULIN (*Bull. de l'Acad. Roy. des Sciences de Belgique*, t. xiii., No. 3).

Conclusion, that it is the remains of neural ridge (crête neural), which explains its relation with the vagus roots and the dorsal branches of the spinal nerves.

12. The Neurenteric Canal of Vertebrates. KUPFFER (*Sitz. d. Gesellsch. f. Morph. u. Phys. Munich*, 1887. III.).

Critical summary of recent observations on development of fishes and amphibia. Conclusion, that the blastopore is the original anus of vertebrates, and that this relation is changed by the caudal extension of the central nervous system, leading to the formation of the neurenteric canal and secondary anus. Probability that a stage existed in which the gut and the neural-canal had a common opening.

13. The Morphological Significance of the Epiphysis (Pineal Gland) in Vertebrata. CH. JULIN, Lille, 1887 (*Rep. Bull. Scient. de Lille*).

PHYSIOLOGY—GENERAL.

14. The effects of Total Anæmia on the Brain and its Diverse parts, studied with the aid of Decapitation followed by transfusions of Blood. HAYEM AND BARBIER (*Arch. de Physiologie norm. et path.*, vol. x. pt. 5, p. 1, *July*, 1887).

A series of experiments in which the carotids of a dog were put into communication with a supply of blood, usually from a living horse, and decapitation of the animal then effected. The transfused blood allowed to circulate either at the moment of decapitation or at varying intervals thereafter. Record of reflex and voluntary movements exhibited by the decapitated head. Their relation to the interval before transfusion commenced, &c.

15. Physiology of the Frog's Brain (*continued*). DR. MAX E. J. SCHRADER (*Pfluger's Archiv*, *July* 26, 1887).

Series of 42 critical experiments testing statements of Goltz and Steiner with regard to the physiology of the frog's brain, and necessitating certain emendations in their views. For example, circumstances are enumerated under which the frog deprived of its cerebral hemispheres behaves like an intact frog, not exhibiting the total want of spontaneity and Will ordinarily ascribed to it. Author comes to general conclusion, that the nervous system

shows a physiological segmentation corresponding to its morphological disposition, each segment having certain functions peculiar to itself to perform.

16. Structure and Functions of the Brain. T. V. Rohon. (*C. Winter, Heidelberg*, 1887. 8vo. 39 pp.)

Sensation.

17. Influence of Trigeminus Stimulation on the Touch and Temperature Sense of the Face. Victor Urbantschitsch (*Pflüger's Archiv f. Physiologie, July* 26, 1887).

Having found that in cases of disease of the middle ear the neighbouring regions of the head are less sensitive on the diseased than they are on the sound side (for instance, the Eustachian tube is less sensitive to the passage of a sound), the author proceeds to investigate the extent of these variations in sensibility, duration of after-sensation, &c.

18. The Perception of the Direction of Sound by means of the Semicircular Canals. W. Preyer (*Pflüger's Archiv, July* 15, 1878).

Impossible to give an outline of this paper within the limits of this index.

19. The Sense of Smell in the Dog. G. J. Romanes (*Revue Scientifique*, 1887, *August* 13).

Experiments to determine the extent of the power in the dog of recognising his master's footsteps from amongst those of other people; odour belonging to the boot rather than to the person; insulation of boot from the ground by brown paper sufficient to mask the scent; anointing boot with oil of anise not sufficient, and so forth.

20. Note on the Specific Energy of the Nerves of Taste. W. H. Howell, Ph.D., and J. H. Kastle, S.B. (*Studies Johns Hopkins' Biol. Lab.*, 1887, *June*, p. 13).

The authors have made use of a substance, para-brom-benzoic sulphinide, which gives on the back of the tongue a pure bitter, and on the front of the tongue a sweet taste. At the tip of the tongue a slight bitter usually precedes the sweet taste. The

stimulation of different sensations in the two regions of the tongue, regarded as a proof of the existence of separate nerves for specific tastes.

Physiological Psychology.

21. Sensation and Movement, Experimental Studies in Psychomechanics. Charles Féré (*Alcan, Paris*, 1887).

Introduction of measurement into domain of psychology. Study by means of dynamograph and plethysmograph of the influence of sensations upon the static force of the individual. Influence of position, gesture, &c., in suggesting ideas. Relation between the idea of movement and movement. On account of their hyper-sensitiveness, hysterical persons are largely made use of in these experiments. Pessimism a result of *dégénérescence* and other philosophical applications, &c.

Reaction Time.

22. Reaction Time for Action and Inhibition, gathered from Observations of Dr. Orschansky, Physiological Society in Berlin (*Archiv f. Anat. u. Phys.*; *Phys. Abth.*, 1887, p. 363).

Hypnotism.

23. Auto-suggestion in Hypnotized Subjects. N. Cybulski (*Phys. Centralbl.*, 1887, *Sept.* 3).

Cortex.

24. The meaning of Brain-fissuring. T. Seitz (*Toeplitz und Deuticke, Vienna*, 1887. 8vo. 67 pp.).

25. Contribution to the Morphology of the Island of Reil. G. A. Gullberg. (*Christiania*, 1887. *Danish.*)

Localisation.

26. The Localization of Brain Disease. Nothnagel (*Biol. Centralbl.*, Bd. vii. *No.* 13, *Sept.* 1).

Historical and critical essay read at the Medical Congress at Wiesbaden. Treats especially of the localisation of visual perceptions, and discusses the possibility of localising the cortical lesions in different portions of the visual area, leading to various forms of disturbance.

27. Eye-movements after Injury to the Nervous System. K. Szigethy (*Orvosi Hetilap*, 1887, *No.* 4, *in Hungarian. Abs. in Phys. Centralbl.* 1887, *Sept.* 3).

A series of experiments upon the movements of the eyes which follow mechanical injury to various parts of the brain in rabbits—particularly the medulla oblongata, mid-brain and cerebellum. Shows that compensatory movements of the eyes when the head is displaced still take place after destruction of the vermis.

Corpus Striatum.

28. On the Functions of the Corpus Striatum, and concerning a Basal Optic Nerve-root. L. Edinger (*Münchener Med. Wochenschr.*, vol. 34, No. 26).

Pituitary Body.

29. A case of Persistence of the Hypophysial Canal (*Anat. Anzeiger, No.* 16, *July* 15, 1887.)

Found p. m. in a girl æt. 14. The canal opened 2 mm. above the upper end of the pharyngeal tonsil (not into the bursa pharyngea). It contained a cord of substance resembling the pituitary body in constitution.

Mid-Brain.

30. The Mutual Relation between the Central Origins of the Eye-muscle Nerves. Nussbaum (*Medizinische Jahrbücher*, 1887).

Correction of Duval's and Laborde's results. Sections of brains of young cats stained by Weigert's method in carmine. Abducens nucleus of each side sends a diffuse bundle of fibres to posterior longitudinal bundle, crossed relation of post. long. bundle with the root fibres of the trochlearis. No crossed relation with the oculo-motorius visible.

Medulla Oblongata.

31. Studies in the Innervation of Respiration (seventh communication). O. Langendorff (*Archiv f. Anat. u. Phys.; Phys. Abth.* 1887, p. 237).

Author formerly showed the existence of spinal reflex centres, and concluded that a single respiratory centre in the sense of Flourens' *nœud vital* does not exist. Contends that the complex

of centres has no anatomical but only a physiological individuality. The article contains a critical *résumé* of recently published experiments and views, and shows how they harmonise with this theory.

32. Facts showing that it is because the Medulla Oblongata is the principal seat of Inhibition of Respiration that it appears to be the principal centre of Respiratory Movement. BROWN-SÉQUARD (*Comptes Rendus Soc. de Biol., Paris*, 1887, *May*).

Contention that there is no such thing in the medulla oblongata as a *nœud vital*, in the sense of a centre liberating respiratory movements. The cessation of respiration which follows injury to the medulla oblongata depends upon irritation of the inhibitory mechanism. Same standstill may be brought about by stimulating the trigeminus, &c., without injuring the medulla. In young animals section of the cervical cord does not prevent thoracic respiration. In some cases inhibition of respiratory movements does not follow injury to the region of the "nœud vital."

33. Where is the Swallowing Reflex released? N. WASSILIEFF (*Zeitsch. f. Biol. München u. Leipzig*, 1887, p. 29, *Sept.*).

Confirmation of the observation, that stimulation of supr. laryngeal nerve induces act of swallowing, while section of the same nerve is without effect. That stimulation of the glosso-pharyngeal nerve has inhibitory effect. Determination of a definite area, in the roof of the rabbit's mouth, touching of which is invariably followed by swallowing. This area loses its irritability when the trigeminus nerve is cut.

34. Origin and Connections in the Medulla Oblongata of the Hypoglossal Nerve. P. D. KOCH (*Copenhagen*, 1887).

CRANIAL NERVES.

35. On the Functions of the Fourth Pair of Nerves. HENRY LEE (*Lancet* 1887, vol. ii. p. 9).

SPINAL CORD.

36. The Posterior Nerve-roots, their Connections in the Cord and Central Prolongation. W. BECHTEREW (*Archiv f. Anat. u. Phys.; Anat. Abth.* 1887, p. 126).

Observations by Weigert's and gold chloride methods of cords of fœtuses and new-born children. Posterior root contains two kinds of fibres acquiring myelin sheaths at different times. Those which develop first are also the larger fibres. Most of them go into the ground region of Burdach's column; a smaller number into the substantia gelatinosa. Of the smaller later-developed fibres the greater number go into the posterior part of the lateral column, a few into subs. gel. Further course throughout the spinal cord followed. Larger fibres pass amongst the cells of Clarke's column. Flechsig's observation, that the fibres from Clarke's column go into lateral cerebellar tract confirmed.

37. Sensory Nerves and Reflex Apparatus of the Spinal Cord (*continued*). K. HÄLLSTÉN (*Archiv f. Anat. u. Phys.; Phys. Abth.*, 1887, p. 306).

Determination of the conditions under which reflex action can be obtained through the spinal cord of the frog. (1) By stimulation of the skin. Attempt at drawing up a scale of stimuli. (2) Secondary stimulation of the cut sciatic by means of a contracting muscle. Reflex cannot be obtained by rheoscopic stimulation with a nerve. (3) Double muscle reflex and reflex routes in cord. Attempt to prove that the impulse has the choice of a higher and a lower path across the cord. Upon the path selected depends the latent period and height of contraction.

SPINAL GANGLIA.

38. The Physiology of the Spinal Ganglia. MAX JOSEPH (*Archiv f. Anat. u. Phys.; Phys. Abth.*, 1887, p. 296).

Experiments after the Wallerian method already recorded in Virchow's *Archiv* (cf. 'BRAIN' for July, this index). The notable result of these experiments is the discovery, that there is a partial degeneration of the portion of nerve attached to the spinal when the root is cut either proximally or distally to the ganglion. Also that when the root is cut proximally to the ganglion a portion of the root stump remains intact. Conclusion, that a certain number of fibres between the periphery and the cord pass through the root ganglion without union with its cells. Criticism of current views as to the constitution of the ganglion.

SPINAL ROOTS.

39. Anatomical Investigation into the Spinal-roots of Man. Dr. ERNST SIEMERLING (*Hirschwald, Berlin*, 1887. 8vo. pp. 32, 2 *large plates*).

A purely anatomical study of the constitution of the nerve-roots, especially with regard to the character of the fibres of which they are composed. The relative numbers of small and large fibres in both roots of each nerve. Their grouping and mode of development of the broad fibres, the largest are found to be those which have the longest extra-spinal course.

Sympathetic System.

40. The Ciliary or Motor-oculi Ganglion and the Ganglion of the Ophthalmicus Profundus in Sharks. J. Beard (*Anat. Anzeiger*, 1887, No. 18, *August 15th*).

The ciliary ganglion is by some regarded as belonging to the sympathetic system, by others it is looked upon as a posterior root ganglion. The object of the present paper is to clear up this uncertainty, which is due to the fact that, during the development of lower vertebrates, the ganglion on the ophthalmicus profundus branch of the fifth is conspicuous, and therefore mistaken for the ciliary, whereas in mammals it is fused with the Gasserian. The ophth. prof. ganglion belongs to the root ganglion series, while the ciliary is a portion of the sympathetic system.

41. Anatomy of Ciliary Ganglion and Vagus Nerve in Selachians. Onodi (*Physiological Society in Berlin*) [*Arch. f. Anat. u. Phys.; Phys. Abth.* 1887, p. 357].

42. Processes of Nerve-cells in Heart-ganglia. Nikita Ladowsky. *Communicated by* Prof. C. Arnstein (*Archiv f. Mikro. Anat.*, 1887, p. 609, pl. xxxviii.).

Gold chloride and formic acid 10%. Frogs and rabbits. Relation of nerve-cells to nerve-fibres on the one side, and muscle-bundle on the other described and figured. Various forms of uni-, bi-, and multipolar-cells, or of the former with spiral fibre. Definite observation of the connection of cell processes with the musculature of the auricle.

43. The Nervous System and Animal Heat. Ch. Richet (*Revue Scientifique*, 1887, *Sept. 17th*). Popular résumé.

44. The Relation of the Brain to the Stomach. Bh. Hlasko (*Karow, Dorpat*, 1887. 8vo. 31 pp.).

45. The Significance of the Nervous System for the Kidneys. E. Sehrwald (*Fischer, Jena*, 1887. 8vo. 88 pp.).

46. Studies in the Central Destination of the Vaso-motor Nerve-routes. K. HELVEG (*Kjobenhavn*, 1887. 8vo. *Danish.*)

HISTOLOGY.

47. Histological Alterations in the Central Nervous System in Experimental Rabies. GOLGI (*Arch. Ital. de Biol.*, vol. viii., fasc. ii., p. 192).

Deals with karyokinetic changes in the nucleus of nervous, epithelial and neuroglial cells, and also appearance in nerve-cells of peculiar nuclear mass.

48. On the Histology and Function of the Mammalian Superior Cervical Ganglion. W. HALE WHITE, M.D. (*Journal of Physiology*, vol. viii. pt. 2, p. 66, pl. iii.).

Account of the variations in size of the superior cervical ganglion in man and comparison with mammalia. Description of its histological elements. Attention particularly called to great frequency in man, and to less degree in monkeys, of pigmentary degeneration and atrophy of its cells. This change regarded as "normal" and unassociated with disease.

49. Degeneration and New Formation of Medullated Nerve-Fibres. GIUSEPPINA CATTANI (*Archivio per le Scienze Medichi, Turin*).

Treats of the histological changes which lead to the first formation of a nerve filament after section from the central system, and of its further acquisition of a medullary sheath.

50. Minute Anatomy of Teleostean Brains. R. FUSARI (*Internat. Monatschr. f. Anat. u. Hist.*, vol. iv., Nos. 7 and 8, p. 275, pl. ix–xi.).

Brains hardened in Müller's fluid and osmic acid. Detailed description of the histological elements occurring in the cerebellum, valvula cerebelli, and optic lobes.

51. Nerves in Epithelium. DR. S. FRENKEL (*Virchow's Archiv*, 1887, *Sept.* 1).

Criticism of observations hitherto recorded. Original observations and comparison of results with gold chloride and osmic acid.

Demonstration of the connection of cells of rete Malpighi with one another by means of filaments (feine fäden). Relation of this intercellular network to the nervous functions of epithelium. Theory with regard to the histological differentiation of epithelial cells into nervous and supporting elements.

52. Golgi's Researches into the Minute Structure of the Central Nervous System. Köllicker (*Anat. Anzeiger*, 1887. No. 15, *July* 1).

Results of examination of certain preparations submitted by Golgi. Controverts Golgi's conclusion, that the protoplasmic processes of the nerve cells are not of a nervous nature. Calls attention to the importance of Golgi's discoveries as to the complicated constitution of the plexus of fine processes and the number of elements entering into it, and confirms his observation of the origin of medullated nerves in the grey matter by direct association in the plexus of protoplasmic processes.

Degeneration.

53. Ascending and Descending Nerve Degeneration. Feodor Krause. *Phys. Society in Berlin* (*Archiv f. Anat. u. Phys.; Phys. Abth.*, 1887, p. 370).

54. Degeneration of the Optic Nerve and Chiasm. Julius Michel (*Bergmann, Wiesbaden*).

Brain Weight.

55. Weight of Pranzini's Brain (*Revue Sci.*, 1887, *Sept.* 5), 1280 grammes.

Circulation.

56. Variations in the Development of the Brain-vessels, their Physiological and Pathogenetic Relation. Dr. L. Löwenfeld (*Archiv f. Psychiatrie*, xvii. pt. 3, p. 819).

Great variation in the relation of arterial calibre to brain-weight. Difference in the calibre of the two carotids, most frequently in favour of the left; possibly associated with the frequent preponderance of the left hemisphere.

METHODS.

57. Methyl-blue Staining as a Histological Method. ARNSTEIN (*Anat. Anzeiger* 1887, No. 17, p. 551, *Aug.* 1).

For nerve tissues; using picrocarmine or picrate of ammonia as a fixing agent.

Current Nerve Anatomy and Physiology.—Titles and Indication of Contents of Papers which have appeared during the Quarter (Michaelmas to Christmas 1887). By ALEX. HILL, M.D., Fellow of Downing College, Cambridge.

ANATOMY.

1. Brain of Man in its intimate relations and connections, BECHTEREW (*Archives Slaves de Biologie*, iv. 1, *July* 1887).

Contains an account of the connections of the various nuclei of grey matter in the cerebral axis, as determined by recent researches.

COMPARATIVE ANATOMY AND MORPHOLOGY.

2. Nervous System and general Morphology and Classification of the proso-branchiate Gasteropods. E. L. BOUVIER (*Annales des Sciences Naturelles*, 1887, vol. iii. pp. 1–336, plates 1 to 14, to be continued).

3. Nervous System of Gasteropods (type Aplysia). LACAZE DUTHIERS (*Comptes rendus Acad. des Sciences, Paris*, Nov. 21, 1887).

4. Electrical Fishes, part 1, Malopterurus electricus. FRITSCH (*Leipsic*, 1887. *Veit & Co.* Folio, 90 pp., 12 plates).

5. The Metamerism of the Head and the Vertebra Theory of the Skull. GEGENBAUR (*Morph. Jahrbuch*, xiii. 1, *Nov.* 1887).

GENERAL PHYSIOLOGY. DUALITY.

6. Duality of the Brain and Spinal Cord, as shown by the fact that Anæsthesia, Hyperæsthesia, Paralysis and various states of Hypo and Hyperthermia due to Organic Lesion of the Cerebro-spinal Centre, can be transferred from one side of the body to the other. BROWN-SÉQUARD (*Comptes rendus Acad. des Sciences, Paris, Oct.* 17, 1887).

First of a proposed series of communications, demonstrating that each half of the brain and spinal cord can serve for all the functions of both sides of the system. For example, section of one side of the base of the brain leads to anæsthesia of the opposite leg and hyperæsthesia of the leg of the same side. Subsequent section of the opposite side of the dorsal cord leads to a reversal of these sensory disturbances. If the internal capsule on one side is cut, followed by section of the opposite side of the dorsal cord, the result is the same as in last experiment. Section of the right cerebral peduncle produces paralysis of the left side. Section of the right half of the medulla, above the crossing of the pyramids, transfers the paralysis to the opposite side. In the frog, section of the left cerebral hemisphere produces loss of power on the right side and increased force on the left. Subsequent section of the right cerebral hemisphere restores the equilibrium. Evidence of the same kind is adduced with regard to body temperature.

7. Researches into the two Fundamental Principles of the doctrines received with regard to Cerebral Duality in voluntary movements. BROWN-SÉQUARD (*C. R. Acad. Sci., Nov.* 7, 1887).

Section of right anterior pyramid, and then stimulation of its fibres, is almost always followed by movement of the right leg. The same movement results if the stimulation is at the level of the decussation. Conclusion, that each motor region acts on both sides of the body, and that each half of the base of the brain conducts fibres from both motor zones.

CEREBRAL PHYSIOLOGY.

8. The Brain and Cerebral Activity from the Psycho-physiological point of view. HERZEN (*Paris, J. B. Baillière*, 1887).

A popular explanation of recent advances in psycho-physics. Starts with the proposition, that thought necessitates chemical action (metabolism) of brain tissue, and that therefore it is a form of motion comparable to muscular action. Infers that every psychic act is a transmission or modification of an external impulse. Illustrates the proposition by experiment. Explains "reaction time." Shows that it is longer with children than with adult, with boys than with girls, but after adolescence less in men than

in women. External exhibition of cerebral activity in increased temperature. Latter part of the book deals with philosophical deductions. Discusses the question of free-will and consciousness, and shows the limits within which they are reconcilable with the mechanical definition of thought as a redirected sensory impulse.

9. Physiological Psychology. SERGI, trans. from Italian into French by MOUTON, revised by the Author (*Paris, Alcan*, 1888).

10. The Motor Functions of the Brain and Cerebral Epilepsy. FRANCK (*Paris*, 1887, 8vo. 571 pp.).

11. The Time it takes to Think. J. McK. CATTELL (*Nineteenth Century, Nov.* 1887).

A popular exposition of the objects and possibilities of time measurements as applied to psychic actions.

REFLEX ACTION—KNEE-JERK.

12. The Variations of the Normal Knee-jerk, and their relation to the Activity of the Central Nervous System. LOMBARD (*American Journal of Psychology*, vol. i. No. 1, pp. 5–71, *Nov.* 1887).

A series of experiments made under favourable conditions as to position of the body and regulation of the force and place of incidence of the blow. Knee-jerk shown to be exceedingly susceptible to alteration in force under the influence of external conditions. Depressed by hunger, fatigue, enervating weather and sleep. Reinforced by any condition which increases the activity of the central nervous system, by irritation of the skin, voluntary action, exciting the attention, music, dreams, &c.

REFLEX ACTION—RESPIRATION.

13. Respiration Centre in Medulla Oblongata and the Conditions of its action. LOEWY (*Berliner Phys. Gesellsch. Archiv f. Anat. u. Phys.* 1887, Hft. v.).

14. Respiratory Reflex from the Nasal Mucous Membrane. SANDMANN (*Berliner Phys. Gesellsch. Arch. f. Anat. u. Phys.* 1887, Hft. v.).

Cortex Anatomy.

15. Morphology of the Island of Reil. Guldberg (*Anat. Anzeiger* ii. 21, *Oct.* 1887).

The island of Reil appears on the outer surface of the lateral ventricle, as an elevation at the bottom of a slight hollowing, at the third month, after the olfactory bulb has made its appearance as a button-like out-growth. Paper contains, in addition to account of development, an account of the form assumed by the island in such mammals as possess it.

Cortex Physiology.

17. Cortex-Field of the Facial, and its Connections in the Dog and Rabbit. Exner and Paneth (*Pflüger's Archiv*, xli. 7 and 8, p. 349, *Nov.* 22, 1887).

Anæsthesia was induced by subcutaneous injection of morphia, followed by intravenous chloral. In one case narcotism was very superficial, and in another case morphia only was used, and yet only crossed action obtained even with the strongest stimulus; same-sided and crossed action at the same time were very rarely obtained. In the dog the area for the facial nerve lies in the gyrus lateral to the sigmoid. It was noticed that stimulation of the dura mater gave rise to a reflex contraction of the orbicularis on the same side. This could be obtained over the whole convexity of the brain, but most easily from the anterior part. The reflex was often induced when breaking the skull. In the rabbit, stimulation of one side of the cortex produced without exception movements of both sides of the face. This was not prevented by section of the corpus callosum, nor by dividing the pons, nor by destroying the opposite cortical centre. Section of the medulla, however, stopped it at once, and it is hence inferred that there is total crossing for the facial nerves in the medulla.

Cortex.

16. The Homology of the Fissura Parieto-occipitalis in Carnivores. Max Flesch (*Leipsic*, 1887, *Engelmann*, 4to, 15 pp., 1 *plate*).

18. Physiology of the Brain. Munk (*Real-encyc. der ges. Heilkunde*).

Contains an account of the physiology of the cortex, expressed in terms consonant with the author's well-known views, and also an account of the bibliography of the subject.

19. Further Minute Analysis by Electric Stimulation of the so-called Motor Region of the Cortex Cerebri in the Monkey (*Macacus sinicus*). CHARLES E. BEEVOR, and VICTOR HORSLEY (*Proceedings of the Royal Society*, xliii., No. 258, *issued Oct.* 1887).

This paper, being but a brief abstract of one about to appear in the Transactions, can hardly be further analysed for this index. The region investigated comprised the gyrus coursing in front of the precentral sulcus for its whole length; the posterior third of the middle frontal convolution; the posterior half of the superior frontal; upper end of the ascending frontal; and the whole of the ascending parietal, except the lower half of its anterior border. Head and eyes are turned to the opposite side on stimulating within the broad zone in front of precentral sulcus, and including the posterior half of the middle and superior frontal convolutions as far as the margin of the hemisphere. Lower limb region includes the posterior fifth of the superior frontal, upper third of the ascending frontal, and the upper third of the ascending parietal. Upper limb region includes the middle of the ascending frontal, reaching into the middle frontal and extending upwards slightly into the superior frontal, and backwards over the lower two-thirds of the ascending parietal as far as the intra-parietal sulcus. An account of the "march," &c., will appear in the full paper.

CORPUS CALLOSUM.

20. Absence of the Corpus Callosum in the Human Brain (*continued*). KAUFMANN (*Arch. f. Psychiatrie*, xix. 1, p. 229, *plate* 2, 1887).

Further details with regard to case already reported (cf. this index for last quarter). Conclusion that the tapetum, which always up to this time has been considered a part of the corpus callosum, has nothing to do with it, but belongs in reality to the superior longitudinal fasciculus. This case is regarded as one in which the corpus callosum has never commenced to develop owing to hydrocephalus internus. Second case in which the corpus callosum was totally destroyed by an embolism originating in an

aneurism of the arteria corporis callosi dextra. Subject was a man 45 years old. There were no opportunities of examining the patient's condition before death. As in the first case the internal capsule revealed no traces of change, even to microscopic examination, as the result of the total destruction of the corpus callosum.

Optic Thalamus and Corpus Striatum.

21. The Four Cerebral Heat Centres. Izaac Ott and William S. Carter (*Therapeutic Gazette, Sept.* 15, 1887).

The skull being trephined, a fine instrument was passed through the brain tissue to the region, puncture of which was desired. The wound was washed out with corrosive sublimate and closed with sutures, the animal being then allowed to run about. Injury to various regions about the optic thalami and corpora striata, but particularly, (1) in front of and beneath the corpus striatum, (2) the parts on the median side of the nodus cursorius, (3) the parts about Schiff's crying centre, (4) the anterior end of the optic thalamus, are followed by a rise in temperature lasting for several days, and not accompanied by increase in respiration, pulse-rate, or blood pressure.

Optic Thalami.

23. The Significance of the Optic Thalami as deduced from Experimental and Pathological Data. Bechterew (*Virchow's Archiv*, vol. 110, part 1, pp. 102–154, *Oct.* 3; *continued in* part 2, pp. 322–366, *Nov.* 1887).

Critical summary. General conclusion, that the optic thalami have a prominent rôle in the reception of sensory impressions, and are the seats of complex reflex actions.

22. The Minute Structure of the Corpus Striatum and Optic Thalamus. Marchi (*Rivista speriment. di Freniatr., etc.*, 1887, xii., p. 285).

The nerve cells in these organs are scattered throughout the ground substance and not grouped, nor are their long axes arranged in any particular direction. In the nucleus caudatus they have a size of 15–20 μ. In the nucleus lenticularis, 30–50 μ. In the thalamus they are somewhat larger. All the cells have a single nerve process and numerous protoplasmic processes. The nerve process either passes into a medullated nerve, or loses its individuality and

breaks up into a plexus; the first type is more common in the optic thalamus, the second in the corpus striatum. Fibres from crus cerebri and from corona radiata go into both ganglia. These results are obtained with Golgi's method (bichromate of potassium and nitrate of silver).

Pineal Gland.

24. Structure of the Pineal Gland. Cionini (*Riv. speriment. di Freniatr. e di Med. leg.*, 1887, xii., p. 364).

The author is unable to find any nervous structure in this body with the exception of its vaso-motor nerves.

Cerebellum.

25. The Nucleus Dentatus of the Cerebellum. Sacozzi (*Rivist. speriment. di Freniatr.*, &c., 1887, xiii., p. 93).

The development of this nucleus increases as we ascend the mammalian scale, it being much more important in man than in animals. Using Golgi's method, the author finds in it cells belonging to each of Golgi's two types, "motor" and "sensory."

26. The Cerebellum of the Frog. Wlassak (*Archiv f. Anat. u. Phys.-Physiol. Abth.* 1887, Supplement, *Dec.* 8).

Brain hardened in sublimate solution, or bichromate of potassium and sulphate of copper. Series of sections made either in paraffin or between layers of celloidin, stained with Weigert's hæmatoxylin or with Gaule's method. Author claims to be able to trace into cerebellum the several sets of fibres which connect it with the rest of the cerebro-spinal axis. (1) Posterior-column fibres without crossing to upper part. (2) Lateral-column fibres, partly crossed and partly uncrossed. Uncrossed to under part, crossed to middle part. (3) Fibres from optic lobes for the most part cross over to opposite side of middle part; a few go to deepest layer of same side. (4) Crossed (outer) arcuate fibres to the middle part of opposite side. (5) Direct (inner) arcuate fibres with posterior column fibres on same side. (6) Fibres from pars peduncularis of optic lobes to deepest part of opposite side. (7) Commissural fibres with the posterior column fibres, uncrossed.

Histologically, the nuclei of the nuclear layer are identical with those of the medullary sheath. The nuclear layer arranges for its breaking up. The axis-cylinder of the nerve goes to a basal

process of a Purkinje cell. The medullary sheath forms its capsule. Both are continued into the molecular layer by the branching processes of the Purkinje cell. There is, in the frog, no limitans interna. Confirms Beevor in all points, but unable to recognise any connections of branching processes of Purkinje cells, or arrangement of nerve fibres indicative of a centripetal connection.

Cranial Nerves—Facial.

28. The Nuclear Origin of the Orbito-Facial. Mendel (*Neur. Centralbl.* No. 23, *Dec.* 1, 1887).

Both in cases of cerebral apoplexy, and also in bulbar-paralysis, the orbicularis palpebrarum commonly escapes, while the other muscles innervated by the facial nerves are paralysed. It is usually stated that the cortical centre for the former muscle lies in the inferior parietal lobule, while the centres for the other muscles are situated in the inferior third of the ascending frontal. Author makes use of Gudden's method. Concludes that in rabbits and guinea-pigs, the orbito-facial has its nuclear origin in the hinder part of the centre for the oculomotorius, which probably also innervates the levator palpebræ superioris.

Cranial Nerves—Third, &c.

27. The Crossed Relation beween the Central Origins of the Eye-Muscle-Nerves. Nussbaum (*Wiener Med. Jahrb.* 1887. II.).

29. Facial Nerve in the Domestic Cat. T. B. Stowell (*Proc. American Phil. Soc.*, xxiv. p. 8, 1887).

Cranial Nerves—Vagus.

30. On the Physiology of the Heart of the Snake. Mills (*Jour. Anat. and Phys.*, vol. xxii. pt. 1, *Oct.* 1887).

In the snake both vagi are effective—stimulation leads afterwards to increased force and frequency of beat, or to the former only, and according to the law of inverse proportion previously announced by the writer. During vagus-arrest the sinus and auricles are inexcitable.

31. Action of the Vagus upon the left side of the Heart. Pawlow (*Archiv f. Anat. u. Phys. Phys. Abth.* 1887, Part 5, Dec. 8).

CRANIAL NERVES—SPINAL ACCESSORY.

32. Origin and Central Course of Nervus Accessorius Willisii. DEES (*Allg. Zeitschr. f. Psychiatrie*, xliii. p. 453, 1887).

Studied by means of a series of sections in human embryo, and also in two rabbits in which the nerve was cut six weeks before death. The nerve in its central course can be recognised by the great size of its fibres (15 μ.). Absence of the nerve fibres was associated with the absence of segmental groups of large multipolar nerve cells (30 to 40 μ.). The cells lie in the medulla in the midst of the anterior horn, and incline outwards as the group descends the cervical cord. At C. 4. they lie on the side of the anterior horn, at C. 6. at the base of the lateral horn. Above, they shade off into the vagus nucleus.

NERVE ENDINGS.

33. Changes in Nerve Endings during Inflammation. GROSSMANN (*Mitth. aus dem Embryol. Inst. Wien. Nov.* 1887).

Experiments carried out on the thick skin of the duck's beak, which, as is well known, is very rich in Herbst and Grandry corpuscles, and Merkel's touch-cells. Inflammation was induced by mechanical destruction of the skin. Consequent changes consisted, for the most part, in transudation between the nerve endings and surrounding tissues, followed by various minute alterations.

34. Motor Nerve-endings as seen in Cross-section and in Section-series. KÜHNE (*Verhandl. des Natur. med. Vereins zu Heidelberg*, vol. iv. p. 1, 1887).

35. The Nerve-endings in the Electric Organ (second article), KRAUSE (*Internat. Monatschrift f. Anat. und Phys.*, vol. iv. 1887).

Continuation of memoir published in the same Journal, vol. iii. p. 385, 1886.

Animals examined—Torpedo ocellata, and also for pseudo-electric organ, Raja asterias and other Selachians. As is well known, the electric organ arises from the modified striated musculus con-

strictor arcuum branchialum superficialis. The paper deals more especially with the minute structure and development of the curved fibres (bogen-fasern). A trace of cross-striation is recognised in them. In the smallest embryos they are merely granular; later on, they exhibit a fine cross striation and also longitudinal fibrillation. Differentiation between nerve fibre and muscle fibre then appears. Ventral half of fibre outgrows dorsal; becomes multinucleated, and expands transversely into a plate; the nerve fibre which enters it ventrally is carried to one side. It is possible to follow the development of the arched fibres from the striated muscle fibrillæ to the complete electric lamellæ of the adult animal.

Degeneration.

36. Secondary Degeneration of the Crus Cerebri. Bechterew (*Arch. f. Psychiatrie*, xix. 1. 1887).

Records the symptoms during life and autopsies of three cases. Among other points of interest the first case exhibited atrophy of the substantia nigra Soemmeringii, which the author associates with atrophy of the corpus striatum. In the third case there was hypertrophy of the substantia nigra, and an unusual development of the corpus striatum.

Regeneration.

37. Lectures on Injuries of the Nerves: delivered at the Roy. Coll. of Surgeons. Bowlby (*The Lancet*, 1887, vol. i. pp. 863, 921, 968, 1021, 1121; vol. ii. p. 53, 99).

Nerve Conduction.

38. Conduction by Nerves in both Directions. Kochs (*Biol. Centralbl.*, *Nov.* 1, 1887).

Criticism of the results said to be obtained by Paul Bert, by grafting the tip of rat's tail into the cellular tissue of its own back. It was stated by Bert that the abnormal tail acquired sensation in the course of some months. Kochs has repeated this experiment upon forty rats, in thirty cases with satisfactory healing and adhesion of the new tail. In no case, however, was there a return of sensation during the eight months that some of the animals were under observation.

NERVE MUSCLE PHYSIOLOGY.

39. The Stimulus Action of Stöhrer's Machine. GRÜTZNER (*Pflüger's Archiv*, xli. 6, *Oct.* 17, 1887).

40. Chemical Nerve-Stimulation and Action of Salts. LOMBURG (*Pflüger's Archiv*, xli. 7 and 8, *Nov.* 22, 1887).

41. The Tiring of Nerves. HERZEN (*Arch. des Sciences phys. et natur.* 1887, *Sept.*).

Finds that, contrary to usual belief, when a muscle has been tetanised for a long time until it no longer reacts, the nerve-endings are functional, although the nerve trunk is tired out.

SYMPATHETIC SYSTEM.

42. The Central Course of Vaso-motor Nerve-paths. HELWEG (*Archiv f. Psychiatrie*, xix. 1, pp. 104–182, 1887).

Studying sections of the cords of lunatics, the author believes he has discovered in the upper part of the cord a triangular tract which is very probably vaso-motor. The triangle lies just at the junction of anterior and lateral columns, with its base on the periphery and the apex reaching half-way to the anterior horn. It stains strongly with carmine. The fibres are exceedingly slender (1·5 to 2 μ.), with the exception of a few scattered large ones. The author traces the triangular tract upwards through the lateral region of the medulla, the "oval" tract of the tegment, the superior olive, the fillet, &c.

43. Action of the Excised Mammalian Heart. WALLER and REID (*Phil. Trans. Royal Society*, 178, pp. 215–256; *issued Oct.* 8, 1887. *Read Dec.* 16, 1886).

Minute analysis of the alteration in form and electro-motor changes in the excised mammalian heart, and comparison of these phenomena with those hitherto obtained for the frog's heart. Spontaneous beats were observed for a much longer period than usually supposed—in the rabbit's heart as long as seventy-two minutes after excision. Owing to the enormous retardation in the action, an analysis of the phases of contraction, impossible in the heart *in situ*, can be made after it is exposed. Conclusion, that the irregularity in the sequence of changes in the contracting

mammalian heart, as compared with the heart of the frog, can only be explained by supposing that, in the former case, conduction occurs along intermuscular nervous channels, in the latter case along muscular channels. With regard to the electro-motor phenomena, for example, it was found that there was not an invariable diphasic variation as in the frog's heart, but that the movements of galvanometer and electrometer indicate a monophasic variation, negativity predominating either at the apex or the base. A monophasic variation shows a simultaneity of action, which is difficult to understand apart from nervous conduction and co-ordination.

44. The Innervation of Blood-vessels. PIETROWSKI (*Centralbl. f. Physiologie*, 1887, p. 454, *Dec.* 10).

Experiments upon the antagonistic effects, upon the vessels of the tongue, of stimulation of the hypoglossal and lingual nerves. Determination of influence of temperature upon the contracting action of former nerve and dilating action of the latter.

45. Action of Sympathetic on Bird's Pupil. JEGOROW (*Pflüger's Archiv*, xli. 7 and 8, p. 326, *Nov.* 22, 1887—*plate* v., *showing dissection*).

In birds, stimulation of the region in which the first cervical ganglion lies, even after death, is followed by alterations in the pupil. Stimulating soon after death, or nearer to the beak, causes contraction. Stimulating some time after death, or on the side away from the beak, causes dilation. Seeing, however, that direct stimulation of the region, or stimulation of the region after the first cervical ganglion has been taken away, produces the same effect, it is clear that the sympathetic system has nothing to do with it. No effect can be obtained after the fifth nerve has been cut at its exit from the temporal bone.

Physiologically, the cervical sympathetic of birds differs altogether from that of mammals, inasmuch as it does not determine dilation of the pupil. The form of the inter-transverse canal varies very much among birds. In the hen it is well closed. In the turkey it consists only of rings.

The sympathetic in birds has nothing to do with movements of the iris, but contains vasomotor fibres for the skin of the neck and head, the conjunctiva and eyeball.

46. On the Action of Muscarin upon the Heart, and on the Electrical Changes in the Non-beating Cardiac Muscle brought about by stimulation of the Inhibitory and Augmentor Nerves. W. H. GASKELL (*Journal of Physiology*, vol. viii. No. 6, p. 404, Dec. 1887).

Introduces a new method into the study of the physiology of the heart, and particularly the study of the action of poisons upon that organ. The author had already found that, if in making a first Stannius section the coronary nerve was left uninjured, stimulation of the vagus nerve during the period of quiescence, although it produced no visible effect, was yet acting upon the heart, for it affected the character of the contractions of the auricle when it again began to beat. He investigates the electric condition of the quiescent auricle, and finds that stimulation of the vagus is accompanied by an electrical change of an opposite sign to that which accompanies contraction. Muscarin is supposed to stop the heart by stimulating the vagus endings, but Gaskell finds that muscarin does not produce this electric change. After muscarin has brought the heart to a standstill stimulating the vagus produces the electric change already noticed, even when a mechanical or chemical stimulus is used. Muscarin therefore depresses the motor activity of the heart. The augmentor (sympathetic) nerves produce an electrical change of the same sign as that which accompanies contraction. The two nerves therefore, when unable to show their action upon the heart by changing its form, still produce opposite electrical changes.

HISTOLOGY.

47. Nervi Nervorum Periphericorum. PRUS (*Archives Slaves de Biologie*, iv., 2 *Sept.* 1887).

Ehrlich discovered that methylene blue, injected into the veins of a living animal, has a great affinity for fine non-medullated nerves, colouring the terminations of such sensory nerves as the gustatory and olfactory; also the nerves of plain and cardiac muscle fibres. The author finds that, when he makes use of this method, filaments appear in the sheaths of nerve trunks, taking an oblique or transverse course with regard to the fibres contained in the nerve. He supposes that the presence of these nervi nervorum accounts for the spots of local tenderness in the course of nerve trunks in neuralgia.

48. Structure of Nerve Fibres. SCHIEFFERDECKER (*Archiv f. Mikrosc. Anat.* xxx. 3, pp. 435–494, 1887, *Oct.* 1).

Boveri having suggested classification of nerves into, (1) *segmented* peripheral medullated, and (2) *unsegmented* peripheral non-medullated and central medullated nerves, Schiefferdecker directs attention to this point, and finds distinction does not hold. Old classification into, (1) medullated, (2) non-medullated. Discovery that axis cylinder is surrounded by an exceedingly delicate elastic "rind." Conclusion, that all medullated nerves, whether central or peripheral, exhibit Lantermann's and Ranvier's segmentation in the living fibre; watery coagulable substance between segments; reagents penetrate between segments; myelin sheath has no proper nuclei; all central fibres supported by glia substance; white peripheral fibres have connective tissue investment of Schwann's sheath, which has its own nuclei and does not vary in thickness at Ranvier's nodes; axis cylinder is uniform in thickness, and not segmented; within its elastic sheath ("rind") is contained very fluid watery albumen, in which it is possible but not probable that fibrillæ lie; when coagulated it shrinks away from the medulla; the periaxial space being filled with lymph, a coagulum layer (gerinnselscheide) breaks off the axis cylinder; Fromann's lines lie in this coagulum layer; it has nothing to do with the axis cylinder rind. Weigert's hæmatoxylin stains different elements according to the chromium salt used in hardening.

HISTOGENESIS.

49. Cell Division in the Central Nervous System. MERK (*Denkschriften der Math. Naturw. Classe der K. Acad. in Wien*, liii. *Also separate Publ. Karl Gerold's Son*, 1887).

After proper preparation, it is easy to recognise in what part of the embryonic nervous system cell-division is occurring by the presence of karyokinetic nuclear figures. Author investigates various parts of nervous system during their development in all classes of vertebrates. For the spinal cord nuclear figures are most commonly recognizable in the lining epithelium, to which cell-division appears to be more or less restricted.

BRAIN-WEIGHT.

50. Gambetta's Brain. RÜDINGER (*Sitz. der K. B. Akad. zu Munchen*, 1887, p. 69).

At the time of Gambetta's death, it went the round of the newspapers that his brain was phenomenally small, 1100 grammes. This is shown to be quite a mistake, Duval's estimate of 1241 grammes being confirmed. It is stated that the third left frontal convolution exhibited an unusual development.

Circulation.

51. Circulation in the Brain. Gärtner & Wagner (*Medic. Wochenschrift*, 1887, parts 19 and 20).

Experiments on curarised dog. Sinus transversus was placed in connection with the vena facialis postica. Quantity of outflowing blood was measured by Ludwig's kymograph. When the blood-pressure was raised by compressing the aorta there was a corresponding increase in the venous blood flowing from the brain. When the vena cava was compressed, the reverse effect was produced. Raising the blood-pressure by asphyxia, strychnia, stimulating a sensory nerve, and other methods, always increases the outflow of blood from the brain. Chloroform dilates the blood-vessels, but the accompanying fall in blood-pressure may reduce the outflow to zero. Morphia has no action. Amyl nitrite dilates the blood-vessels. The onset of an epileptic attack is marked by a great acceleration of the cerebral circulation.

Literature.

52. Supplement to the Account of Acquisitions to the Anatomy of the Central Nervous System. Edinger (*Schmidt's Jahrb. der Ges. Med.* vol. ccxv., 115).

The Metamerism of the Head and the Vertebrate Theory of the Skull.—C. Gegenbaur (*Morphologisches Jahrbuch*, vol. xiii., part i., pp. 1–114, Nov. 1887).

In the index of papers published during the last quarter will be found the title of this most important article by Gegenbaur, which appears in the current number of the *Morphologisches Jahrbuch.* It was impossible to include amongst the Abstracts an outline of this article, for dealing as it does with one of the most abstruse problems in morphology, and attacking this problem from its most technical side, it is one long closely-reasoned argument. The subject, however, is of great importance to the

neurologist; for no one can hope to understand the constitution of the central nervous system without taking its metamerism into account; and although it is impossible to give an abstract of Gegenbaur's paper, for it does not contain a word which could be left out without detracting from the lucidity and force of the argument, it may be possible, from the great quantity of material which is here collected, to construct a less technical account, which will convey to the reader, who is not by profession a morphologist, an idea of the present position of the subject.

It will be remembered that it was Goethe who recognised, with the instinct of a great poet for nature's harmonies, that the elements used in the construction of the skull are the same which in the trunk enter into the formation of the vertebral column. In Oken's hands, the theory received a scientific form. Huxley, by his researches into the minutely discrete elements of the skull of the bony fishes, carried the problem as far as it was possible to carry it by an examination of the hard parts of the head. It was a question, up to this time, of the homology of the skull with the vertebral column.

With the publication of Gegenbaur's researches into the arrangement of the nerves of the head in Hexanchus, the question enters upon a new and greatly extended phase; for Gegenbaur shows that it is not merely a matter affecting the bony elements of the skull, but a question as to the segmentation of the whole head, which can be best studied in cartilaginous fishes, more especially with regard to the arrangement of the cranial nerves. This is the form in which the problem has for nearly twenty years occupied the attention and tested the ingenuity of morphologists. There can be no doubt that the vertebrate head, complex and heterogeneous as it now is, has been gradually evolved from the anterior end of the body of an animal consisting of similar segments. The elements which enter into its formation can be classified, according to their function, in each single simple segment or metamer of the body of the vertebrate ancestor. The problems to be solved are, (1) the fundamental constitution of a metamer; (2) the number of such metamers entering into the formation of the head. The solutions of these problems are to be found in the arrangement of the elements of the head in the lowest vertebrates, and in the embryos of those higher in the scale. In studying the former, allowance must be made for digression from the primitive type; in the latter, for abbreviation of ancestral history. In both directions a vast amount of work has been done, and naturally the views of those

most interested in the problem have become, with increased knowledge and thought, further and further elaborated, difficult to explain and difficult to reconcile.

In this masterly paper Gegenbaur does not attempt to add anything to our knowledge, but, to use a commercial phrase, takes stock of the additions and inferences of recent years, and subjects them to a searching criticism. Many a piece of fine-spun inference is, to our thinking, crushed by his titanic blows, and little more than the framework of the theory escapes annihilation.

For a long time the tendency has been to increase the number of metamers fused in the head. From the four vertebræ at one time supposed to enter into the constitution of the skull, the number of head-segments has been increased to nine or even more, and this because morphologists have formed a conception of each segment as consisting of a dorsal portion or somite, including a piece of the bas-cranial axis and brain case, with its musculature and nerve; and a ventral portion depending from this as a hoop of bone or cartilage, with its muscles and nerves. Between each two visceral arches is a cleft, the visceral (or gill) cleft, and at the upper end of the cleft, perhaps formed from it, a sense organ with its nerve. All the visceral arches supplied by cranial nerves are included in the head, and regarded as presupposing segments of its dorsal part. The face in front of the mouth is also supposed to be formed of visceral arches, which again pre-suppose a segmentation of the anterior portion of the head. Indeed Dohrn goes so far as to regard the mouth itself as due to the coalescence in the median line of two visceral clefts.

It is against this extreme view that Gegenbaur's criticisms are levelled. If we may reduce his detailed reasoning to general terms, the argument runs thus:—The visceral arches are for the support of the pharynx, the gill clefts for the escape of water entering the mouth; there being no reason to suppose that the mouth has shifted backwards, the presence of visceral arches *in front* of it would be meaningless. Again, while the second somite obviously belongs to the mandible (first visceral arch), and the third somite to the hyoid bone, the fourth somite gives rise to no musculature, and its relation to a visceral arch is uncertain. We have no evidence that the posterior dorsal segments are congruent in their formation with the posterior visceral arches. The number of gill clefts in adult Selachians is subject to variation, and there are other indications of fusion and subsequent redivision, on

account of which the visceral arches lose all value in determining the segmentation of the posterior part of the head. In the same way the branches of the vagus nerve which supply them are not primitively independent, but are formed by the splitting of an originally single nerve. Before, however, discussing Gegenbaur's conclusions, it would be well to give an account of that part of the evidence most interesting to neurologists, derived from the disposition of the cranial nerves.

1. *Olfactory.*—Marshall has attempted to bring this into the category of "segmental nerves," and regards the nose as a "gill cleft." In this he is followed by Beard, who, however, considers the nose as not itself a modified gill cleft, but as the sense organ developed in connection with a pre-existing cleft. It will be readily understood from what we have already stated with regard to Gegenbaur's views that he ridicules the gill-cleft idea. He discusses seriatim the reasons adduced in proof of the segmental character of this nerve, and finds that they have none of them anything to do with metamerism.

2. *Optic.*—This nerve hardly comes into the discussion. Gegenbaur regards it as like the olfactory outside the pale of segmental nerves.

3. *Oculomotor.*—This Gegenbaur considers as clearly proved to belong to the first somite, a somite, however, which since it has no visceral arch is not an independent head segment. Van Wijhe has done important work in showing that the ciliary ganglion is not, as Krause supposed, the ganglion of the third nerve, but belongs to the ramus ophthalmicus profundus (nasal), *plus* a sympathetic ganglion. The ramus ophthalmicus profundus he looks upon, however, as originally an independent nerve and not a branch of the Trigeminus. Gegenbaur cannot regard this as proved.

4. *Trochlearis.*—This is of all nerves the most difficult to account for. In mammals a purely motor nerve, it gives in selachians also a sensory branch. Ontogeny has brought no new facts to light with regard to this nerve. Van Wijhe shows, however, that the muscle which it supplies is developed from the second somite. He concludes, that it is a ventral nerve-root of a segment for which the ramus ophthalmicus profundus is a dorsal root. Considering, however, the peculiarity of its origin, in which respect it has all the characters of a dorsal root, Gegenbaur finds it very difficult to subscribe to Van Wijhe's view.

5. *Trigeminal.*—Marshall and Spencer have shown that the

superior maxillary nerve is a branch of the inferior maxillary, just as the upper jaw which it supplies is in origin an outgrowth from the lower. Gegenbaur looks upon the ophthalmic division of the fifth as the ramus dorsalis of this segment. He considers the fifth nerve as a single nerve appertaining to the first complete cranial metamer.

6. *Abducens.*—Its development shows that this nerve belongs to the third somite, to which also the second visceral arch or hyoid bone answers. Connections with other nerves have not been observed, and we find ourselves in the same case as with the oculomotor and trochlearis. It is interesting to notice that, although no one can suppose that it belongs to more than one segment, the sixth nerve arises by several roots, a character which we may hence infer is without metameric significance. Both Marshall and Van Wijhe look upon the abducens as an anterior root of the facial, but its place of origin proves that it and the trochlearis cannot have the same morphological value.

7. *Facial and Auditory.*—There is no doubt about the origin of these two nerves from a primitively single trunk. The double nature of the nerve is however regarded by several anatomists—as Wiedersheim, Van Wijhe, and Beard, as proving the existence of two segments; but all that ontogeny has done is to show that no second ventral branch exists, and that there is consequently no evidence of two ventral metameric portions.

8. *Glosso-pharyngeal.*—This is the only nerve with regard to which there is no controversy. It courses along the arch comprising the hyoid bone. That no ventral root is known for this segment is in accordance with the fact observed by Van Wijhe, that its somite is not developed into muscle.

9. *Vagus.*—While Gegenbaur looks upon this as typically a segmental nerve, he does not consider that we have any evidence as to the relation to the head of the gill arches which it supplies. The study of its development teaches us, that in origin it is a single outgrowth, which only subsequently becomes divided into several roots.

10. *Hypoglossus.*—This nerve Gegenbaur regards as the ventral root or roots of the vagus. He also looks upon the branches, which have been described as ventral roots of the vagus, as belonging to the hypoglossal.

In this Journal it would be perhaps out of place to analyse the evidence brought to bear upon the question from a study of the development and adult constitution of the bones and muscles of

the head. Enough has been said to show that this paper is a protest against the treatment of the subject which has recently come into vogue. Gegenbaur considers that it is impossible to reconstruct the head of a series of similar segments composed of parts of equal value. The formation of the visceral arches and the consolidation of the cranium are events belonging to different epochs. Only two segments, the mandibular and hyoid, present an equal development throughout, the rest have undergone fusions and subsequent divisions by which the relation to one another of the somitic or cranial portion and the branchial portion has been permanently dislocated.

The morphological value of the head metamers is not the same for all. The first six are palingenetic; but even of these, four to six are rudimentary in their dorsal sections; seven to nine are cainogenetic; indeed the original head segments have probably fallen away, and their place has been taken by trunk segments. The lower branches of the vagus nerve belong to these secondary segments.

It is very interesting to follow the author of this paper, in his description of the changes by which he imagines the permanent head has been evolved. Amphioxus, he thinks, although not perhaps in the direct vertebrate stem, affords valuable data as to the structure of the animals which preceded the Craniota. It indicates that they possessed more somites and gills, and a more extended pharynx, than the Craniota. The specialisation and increase in the development of the sense organs led to the formation of a brain. This again necessitated the consolidation and fixation of the anterior end of the body. The result was that of the somatic muscles, only those which found some new work to do —i.e. the movement of the eyes—were any longer of use; the rest disappeared. The head became more compact. The anterior gill arches increased in size; the posterior ones atrophied, and their place was taken by trunk segments. As the eyes grew, the animal became more effective. It adopted a better means of obtaining food, and turned one of its gill-arches into a jaw.

The author does not tabulate his conclusions (a table would have enabled one more quickly to grasp his results), but from what has been already said it will be seen that he arranges the head segments thus:—

The first somite with the oculomotorius and ramus ophthalmicus profundus do not constitute a metamer, since no ventral element belongs to it.

The first metamer comprises the second somite, the jaw, the trochlearis, the inferior maxillary, and its branch, the superior maxillary, to the upper jaw.

The second metamer, the third and part of the fourth somite, the hyoid bone, the abducens and facial and auditory nerves.

The third metamer presents a somite (the fifth), which gives rise to no musculature, and has consequently no nerve; to its visceral arch (the first permanent gill arch) belongs the glosso-pharyngeal nerve.

Of the remaining metamers, only the first comprises a head-somite (which gives rise to no musculature), the others are defective dorsally. The vagus nerve is common to them all.

As we remarked at the commencement, the paper is one which might be translated with profit, but which will not bear abstracting, and we can only hope that, by attempting to give an outline of its general tenor, we have not distorted its meaning by suppressing the subtleties and qualifications inherent to the subject.

ALEX. HILL, M.D.

Discussion at the International Medical Congress, 1887, on the Relationship of Syphilis to General Paralysis of the Insane.—(*The American Journal of Insanity*, October 1887, p. 275, *et seq.*)

The relation of Syphilis to General Paralysis of the Insane is a subject of the greatest theoretical interest and practical importance to the alienist. The subject was pretty fully discussed at the recent meeting of the International Medical Congress, and as the 'American Journal of Insanity,' in which a full abstract of the debate appears, is not readily accessible to most British readers, it may not be amiss to give a condensed statement of the opinions which were expressed by the chief speakers who took part in the debate.

Dr. SAVAGE (London) who opened the discussion, described several groups of cases in which syphilis is related to general paralysis. In the first group, he placed cases of syphilis of long standing which have been followed by general paralysis with acute symptoms. He stated that he had seen several cases of this kind in which, after seventeen, eighteen, or even thirty years' standing of syphilis, there had been a sudden outbreak of general paralysis, which in several cases had ended very rapidly. "Even in the best cases," he said, "I have been able to exclude evidences

of intemperance, or of other exciting causes: in fact, I was able to exclude almost every other cause of general paralysis with which I was familiar."

In another group, he placed ordinary cases of general paralysis of the insane, which run a perfectly ordinary course, with no longer and no more frequent remissions, with fits and sometimes without fits—in fact, in no way separable from ordinary general paralysis, with very definite histories of syphilis.

In a third group, of which he had seen many examples, were included cases in which, after a well-marked history of primary syphilis, symptoms due to a local syphilitic nerve-lesion—such as paralysis of the muscles supplied by the third nerve, monoplegia, hemiplegia, simple aphasia, aphasia with hemiplegia, &c., appear,—are treated, in some cases are cured, by antisyphilitic treatment, and sooner or later become weak-minded, and develop symptoms of general paralysis of the insane.

In a fourth group, cases of general paralysis starting in the spinal cord. With regard to these cases, he said: "In introducing the subject, I would say that there are a number of writers who recognise the fact, that those patients who suffer from locomotor ataxy—if we call that a disease, and not an assemblage of symptoms—the largest of them have got some syphilitic history; and one finds that, among general paralysis of the insane, a very fair proportion of them begin with locomotor ataxy. Well of course there are two or three groups: those who begin with locomotor ataxy, and develop into general paralysis of the insane; and those who begin with the ataxy and the general paralysis develop about the same time. Besides these, one gets more frequently, I think, cases of general paralysis in which syphilitic histories are present, and what I have called spastic symptoms, more for convenience, and in contrast to, the ataxic symptoms, and implying a general likeness.

"My experience is that general paralysis which depends upon syphilis, may run a somewhat unusual course, in so far as the remissions may be more frequent and the remissions may be more prolonged. . . . My own experience is, that I have seen only one thoroughly well-marked case of general paralysis of the insane appear to get well; that is, he was able to administer his affairs for some years, but he died ultimately of some obscure nervous disease under Dr. Ferrier. Unfortunately, no post-mortem was allowed, and I am still inclined to think that, though apparently cured, it was only a case of syphilitic general paralysis, in which

there was a prolonged remission. My belief is, that general paralysis of the insane is a degeneration which may be set up by many causes. There is no special form of general paralysis which depends upon syphilitic changes; we may have quite a large number of cases of general paralysis in which there is a syphilitic history, yet not every case of general paralysis with syphilitic history depends upon syphilis. But I feel quite sure that there are one or two definite groups in which there is no doubt whatever, and my belief is that the most important one is that in which local lesions of the nervous centres are present. I fear I am not able to say what these lesions may be. In speaking of the pathological changes yesterday, I said there were no gummatous changes found. I believe, if any are certainly dependent upon syphilis, it is those cases where the symptoms are first of local nerve-cranial lesion, followed by more or less recovery, followed later by degeneration; and my belief is that, in the majority of these cases, the old lesion has formed a focus of degeneration. The only other group generally syphilitic are those ataxic cases in which the ataxic precedes the paralytic symptoms by some considerable time."

Dr. MICKLE (London) expressed general concurrence with the views of Dr. Savage. He had seen few cases of the first group mentioned by Dr. Savage; but he had often seen cases of the other two groups mentioned by Dr. Savage, in which the syphilis was a cause of general paralysis.

Dr. LANGDON DOWN (London) said: "I have had a great deal of experience in London hospitals of locomotor-ataxy cases, and I think they are nearly all of a syphilitic character; that they all respond to antisyphilitic treatment; and that they are, as a rule, syphilitic. I am very strongly inclined to the belief in the syphilitic nature of locomotor ataxy. Is there any connection between locomotor ataxy and general paralysis of the brain? I have had the opportunity of following out several cases very closely." The particulars of a case of locomotor ataxy, in which, after ten years, well-marked fatal general paralysis developed, were then related.

Dr. YELLOWLEES (Glasgow) said: "We are all apt to have, and do have constantly, cases of general paralysis with constitutional syphilis passing its ordinary cause, and without anything exceptional whatsoever about it. I think we want to be careful in our deductions to the effect of syphilis in the history of the disease. They unquestionably coincide and concur in the same individual, without the cerebral paralysis being at all perceptibly

modified thereby. That being so, I think we want to be careful about our deductions. I concur, however, emphatically with what Dr. Down and Dr. Savage have said about the probable syphilitic origin of those cases which begin with spinal symptoms. The result seems to be that in cases of general paralysis occurring in patients with a history of constitutional syphilis, however, that we have the disease modified to a greater or local extent by local paralysis—and by a greater tendency to local paralysis than in the ordinary cases, and that this is the whole of the matter; at least I don't know that our present knowledge gives us farther light than this gives us."

Dr. Nichols (New York) said: "In my experience I have only been able to trace the existence of syphilitic disease or hereditary taint in about half the cases, and allowing for some uncertainty, in other cases it has been my opinion that about two-thirds of our cases have had or may have had syphilis. In respect to the remaining third it seems to me that it is pretty clear that they have not. I sympathise with what fell from Dr. Yellowlees. I have really doubted whether syphilis was an essential cause of general paralysis of the insane. It seems to me that those cases that I have not been able to trace to syphilis run their course more regularly than those that I cannot. I have never been able to benefit a patient that has not had syphilis by antisyphilitic treatment, but I have retarded the disease in many cases in which I knew the patient had had syphilis. . . . I have supposed that excessive venery, excessive intellectual labour, and loss of sleep were the most efficient causes of general paralysis of the insane. These causes, it seems to me, will produce his form of mental disease independently of syphilis. It has seemed and does seem to me that syphilis is not an essential cause of general paralysis of the insane."

Dr. Spitzka (New York) agreed with every material point advanced by Dr. Savage, and said: "There does seem to me to exist a group of cases in which, with a background of progressive dementia, the suddenness of development of certain motor symptoms and the suddenness of their disappearance, as also a peculiar lacunar disturbance of the memory, separates them from ordinary paretic dementia. Pathologically I think they are characterised by two sets of changes; first, a peculiar form of endymal granulation in the ventricles. In ordinary hydrocephalus, paretic dementia, and epilepsy the granulations are warty. In syphilitic dementia I have found the reticular like the ridges of

butter left in separating the halves of a sandwich. In the central tubular grey, particularly of the mesencephalon—and this accounts for the oculo-motor troubles—small hæmorrhages are common. I have brought with me, and have shown to some of the members present, specimens in which the exact localisation of a combined internal rectus and accommodation paralysis was possible. Both anatomically and clinically this disorder differed from ordinary paretic dementia of syphilitic origin. Amongst the accessory causes of the latter complaint tobacco is a most important one. The constantly growing evil seen in large capitals is the habit of imperfect coitus indulged in for the purpose of prevention of conception. This has a most deleterious effect upon the spinal apparatus. Another is the vicious habit indulged in by those who are losing sexual power. These certainly are causes of locomotor ataxy. I believe that wine, women, and worry are the most potent factors in causing general paralysis."

Dr. Hughes (St. Louis, Mi.) said: "I have a conviction that there is something—a vascular condition, a microscopic vascular condition, which will ultimately be discovered to have preceded the coarser structural microscopic changes which we see in the arachnoid vessels in general paralysis. . . . As you know, we are now at a stage in regard to general paralysis of the insane—we stand much as our ancestors did in regard to phthisis pulmonalis, and in regard to Bright's disease and other affections which have been closed as incurable, but which may yet be proved to be curable in certain stages and under certain circumstances. I believe general paralysis will yet be classified as a form of curable disease if taken early enough for treatment, not after they are sent to asylums, but those cases which come under the notice of the neurologist and psychiatrist, before they become fit subjects for the asylum. I have a conviction that the paralytic stage is one of capillary hyperæmia, and the system to be gotten at is one which our pathologists must study for us."

Dr. Savage: "As to the question of vascular changes, I have quite thought with Dr. Hughes that if any cure is to take place it is to be when the case is taken early. The longer we have experience with the disease, and the more we get the early history, the length of time which the prodromal symptoms have existed is extended. I have within the last five or six years made it a rule when a patient became certainly and undoubtedly general paralytic, and was recognised as such by his friends, then to issue a form of question as to the very earliest changes in character, in hand-

writing, in vision of one kind or another; and in a very large number of cases, eight, nine, ten, or even twelve years before the patient became fit for certificates, there were signs of the disease, but I think that not in one case out of ten thousand would we be able to persuade the patient that he required rest or treatment. Especially in some cases with syphilis, I think early treatment will do good; but I am afraid the time is very far distant when we shall be able to persuade those that break down with general paralysis that they required treatment years before."

Dr. HURD (Galesborough, Ill.): "I would like to ask Dr. Savage a brief practical question. Granting that we have cases of general paresis of unmistakably syphilitic origin—I think we all agree that we do find them—is there any reason to anticipate benefit from antisyphilitic treatment?"

Dr. SAVAGE: "Some patients, undoubtedly, with syphilitic histories who are suffering from general paralysis of the insane, rapidly pass under such treatment into conditions of temporary remission. The only case of prolonged remission that I have seen, or nearly all the cases that have syphilis and have prolonged remissions, were treated very definitely antisyphilitically. . . . The consensus of opinion seems to be that I was right in saying that some cases of general paralysis undoubtedly come from syphilis. . . . We are cautioned by Dr. Yellowlees, and I take the occasion of saying I agree with him when he says we have reached in this syphilitic tide of pathology the highest point, and that it is now time to make a short line. I think the border line that Dr. Spitzka has referred to, the cases of syphilitic dementia are the ones about which we shall always have the greatest difficulty. I do not pretend for a moment to be able to say when a case is brought before me: 'this is a case of syphilitic dementia that will live for years, and which might fairly be called dementia organica.' I do not pretend to be able to say that, nor to say, this demented case will not take on a more acute process and end as a general paralytic. I do not believe there are any defining lines in these cases. I think we are perhaps too prone to be definite in these matters, especially since we know comparatively so little of the relationship between syphilitic dementia and syphilitic general paralysis. Accept dementia in these cases, and if they end in this way, let us say they are cases of general paralysis of a demented type, starting with syphilis."

BYROM BRAMWELL, M.D.

Cousot on Periodic Paralysis (*Revue de Méd.* March, 1887, pp. 190–203).—Among the paralyses that accompany or follow upon acute disease, is a group that is attached to malaria, which have been called intermittent paralyses. Comparable to these in some points, though fundamentally distinct, is a group of transitory paralyses which do not follow any acute disease or come on at quite regular intervals, of which Dr. Cousot has observed five cases, and collected two others, and which he wishes to call periodic spinal paralyses. Dr. Cousot's five cases were all members of one family, viz. the mother, two of her four sons, and two of her four daughters. The family was in all essentials healthy. The parents died of small-pox; all the family were, as a rule, in good health. The four paralytic children were a good deal smaller in stature than the rest. The second son, Henri, was the patient selected for the closest examination. He was a man, æt. 34, and since he was 14 years old, had had three attacks of transitory paralysis. They became rather more frequent until he was æt. 20, and then remained stationary. He had typhoid fever, æt. 10, but no other serious illness. He was only 5 ft. high, but strong and athletic. When his attacks of paralysis were coming on, he first felt his knees weak, then his other joints; he was restless and tried to move about; there was no pain. For two or three hours the paresis increased, after nine or ten hours it had gradually disappeared. He remained fully conscious, and had no discomfort beyond a slight headache. He had been carefully put through all electrical tests for his muscular action and sensibility before an attack, and the results then obtained were decisive and normal; the fields of vision and colour were also normal. During an attack, whilst all his body and limbs, but not his head, was in a state of flaccid and complete paralysis, both muscles and nerves gave no reaction at all to galvanic or faradic excitation. As the paralysis began to go off, the reaction of the nerves to a galvanic current was the first normal symptom to appear.

Sensibility remained throughout perfect in all forms during the attacks; but the reflexes, except the papillary, disappeared; the sphincters were never relaxed. In his abnormal state he was covered all over with a peculiar oily sweat, a kind of seborrhœa. The temperature in the mouth was 99·2. On seven consecutive days these attacks were watched and tested; they lasted on an average about ten hours, and as a rule began about midnight, or in the early morning hours, and lasted till about midday and as

late as 2 P.M. Once the fingers were movable, but, as a rule, the paralysis at the culminating point of the attack was complete in the arms, body, and legs, and the sensation perfect.

In the mother of the family similar symptoms had shown themselves about every fortnight. In François, æt. 36, her eldest son, they were of the same type as in Henri, but longer and more severe.

In Emérance, the eldest daughter, æt. 32, the attacks appeared when she was 10 years old, and disappeared after the birth of her first child.

In Josephine, her sister, æt. 21, the symptoms came on when she was 10 or 11 years old, and recurred nearly every week. In her, as in her sister, there was never any menstrual abnormality.

The cases are to some extent analogous to a case of Westphal's.[1] A child, æt. 5, had scarlatina, with perhaps some nephritis, and a month later had an attack of temporary paralysis with thirst, sweating, and loss of electrical reaction in the muscles. These attacks recurred at first about every month, and afterwards more frequently. There was no history of nervous disease in the family; the child seemed in all other respects in very good health. He came into hospital under Westphal's care when he was 12 years old. Many attacks were watched, and one may be taken as typical. In the afternoon he said his right knee was giving way, and fell on the floor; both legs were weak; both arms grew equally weak. There was much thirst, and frequent desire to micturate without immediate power to do so. By midnight the paralysis was complete. When the patient was laid on his back, he could not move any limb; there was no contracture; there was no plantar reflex, but the knee-jerk and cremasteric and abdominal reflexes were normal. Sensation and consciousness were natural; the muscles of the head, face, and eyes, natural also. Strong faradic stimulation of the nerves gave slight results; the trunk of the peroneal nerve could not be stimulated at all, and the muscles which it supplies gave no direct reaction to electricity. Next morning, the reaction to galvanism and faradism in the arms was much better, though still slight in the legs. In the evening, the patient could walk, though he was weak. Subsequently, similar attacks occurred nearly every week. If the boy was warned by some prodromata of pain or weakness in the day, he could stop or lessen his attacks at night.

Hartwig[2] relates a somewhat more complicated case of temporary

[1] 'Berl. Klin. Woch., 1885, p. 489–509.

[2] 'Dissertation,' Halle, 1874.

paralysis in a man who had had tertian ague five years before. He also lost nearly all reaction to electricity during the attack, and the muscles of the head were unaffected. He improved much by treatment with quinine.

These five cases had no present signs of intermittent fever about them, no shivering, pyrexia or enlargement of the spleen, and in four of them there was no history of it. Westphal frankly admits that he cannot interpret his facts, and does not know where to place his case among nervous diseases. There would be a strong inclination to put down the cases to malingering, if it were not for the undoubted change in the reflexes and the electrical reactions. It may be correct to call it a paralysis of inhibition. But it would have to be remembered that that goes a very little way towards explanation when we know of no cause for the inhibition.

In one or two cases the tongue was a little affected, and in Hartwig's case the actions of coughing and sneezing. This would lead us to suppose that the exciting cause extended up into the medulla; otherwise the cases were purely spinal.

The only case that would submit to treatment was the one first described (Henri ——). Quinine in large doses was first used with doubtful benefit; then strychnine, bromides and ergotin without success. Constant currents along the spine, and faradisation of the muscles seemed to do slight harm. The actual cautery and potassium iodide led to no change.

The process of treatment by suggestion is what Dr. Cousot would have preferred to use, but unfortunately his patient was not amenable to hypnotic influence.

A. T. MYERS, M.D. Cantab.

Gerhardt on the Diseases of the Cerebral Arteries—(*Berliner Klinische Wochenschrift*, 1887).—In a Lecture delivered at the Berlin Medical Society (February 3rd, 1887), after commenting on the special functional adaptations of the cerebral vascular system, the relations of pressure in the cranial cavity, the part played by the arteries in the production of brain disease, and the government by the brain of its own vessels, the author discusses certain affections of the latter, *i.e.* aneurisms, rupture, emboli, and thromboses. Respecting the causation of aneurisms, he inclines to think injury of more frequency than is generally supposed; alcohol and syphilis, but especially the latter, to be powerful factors. The basilar, and next the

middle cerebral, are the arteries usually affected by aneurism, whilst the vertebral also suffer frequently. In three of the latter cases, acute bulbar paralysis occurred with uni-orbi-lateral paralysis. The symptoms are marked by a more sudden advent, and periodical retrogression, than is found in softening or hæmorrhage. In these cases a systolic bruit can be heard on cranial auscultation. The latter should be used more frequently, in Dr. Gerhardt's opinion.

Emboli are found on the left side in the proportion of six, to two on the right. The left vertebral offers especial facilities to emboli, since its opening into the basilar is more constricted than the rest of its lumen, whilst from this it follows that the basilar is not often affected. In only about five per cent. of the cases of cardiac valvular diseases do emboli occur; those from the valves chiefly pass into the left middle cerebral, those derived from the walls of the heart, into the right.

In women, emboli are found in the proportion of two-thirds to one-third in men. In only one-third of the cases does complete unconsciousness occur; where the latter is prolonged, it points to hæmorrhage.

Hæmorrhage results from increased blood-pressure and diseased vessels as exciting and predisposing causes. G. considers miliary aneurisms as frequent, but by no means the sole occasions of the rupture. In certainly twenty per cent. of the cases syphilis was a predisposing element.

G. has found Prevost's statements with regard to symptoms, correct in over two-thirds of his cases, *i.e.* the eyes turned to the sound side (or towards the injured hemisphere), whilst in bulbar lesions, to the paralysed side; the reverse when the extremities are convulsed. Excluding tobacco and alcohol, syphilis can account for most of the diseases of the cerebral arteries, especially those of bulb, therefore anti-syphilitic treatment in nearly all cases may be tried.

E. BIRT.

Rybalkin on Paramyoclonus Multiplex.—(*Mierzejewski's Vestnik Klinitcheskoi i Sudebnoi Psikhiatrii*, 1887, vol. i. pp. 223–235).—Dr. Jakov V. Rybalkin, of St. Petersburg, details the fourth Russian (and the thirteenth international) case of a curious and still rather obscure neurosis, described in 1881 by Friedreich under the name of "paramyoclonus multiplex" (cf. 'BRAIN,' 1882, Vol. XVII. p. 136). The patient, a weak carpenter, aged 15, who

had badly fallen from a height (14 feet) about four years before, and who for the last three years had been daily engaged in heavy work (sawing boards three inches thick), that induced cramps in his limbs, passed through two distinct attacks, the first of which lasted five weeks, and the second eight, and which were separated by an entirely free interval of about ten months. The symptoms (as observed during the second attack) consisted in clonic contractions (*a*) of some symmetrical muscles of the upper limbs (Mus. biceps, triceps, pectorales major and minor, and supinator longus); (*b*) of those of the right lower extremity (Mus. vastus externus and internus, rectus femoris, biceps and semimembranosus); (*c*) (occasionally) of those of the neck and body (Mus. sternocleidomastoideus, cucullaris, rectus abdominis, and long spinal muscles); (*d*) of the laryngeal muscles (closing the rima glottidis); and (*e*) (occasionally) of the masticatory muscles. The number of individual contractions usually varied from forty to sixty and more a minute; but sometimes the movements followed one another so rapidly that a tetanoid spasm of two or three seconds' duration resulted. [Tonic contractions, however, which were observed in the patients of Marie Bekhtereff (cf. the 'London Medical Record,' August 1887, p. 337), did not occur in the author's case.] The convulsions were kept within their usual moderate limits solely by the patient's incessant voluntary efforts. When he let his limbs alone (uncontrolled), usual slight twitchings of his limbs at once gave place to rapid jerk-like, alternating flexions and extensions of the forearms, adductions of the arms, &c., the convulsions swiftly spreading over the muscles of the neck and body. The contractions remained absolutely unaltered during voluntary movements, but they distinctly decreased when the patient assumed a recumbent position, or when his attention was diverted by reading or conversation; they ceased altogether during sleep (as in all cases, except Bekhtereff's) and strongly increased from emotion, titillation of his soles, pricking with a pin, faradic shock, stripping his body bare, pressure on the muscles affected, &c. The contractions of the laryngeal muscles were invariably intensified from reading aloud, speaking, &c., the patient suddenly losing his voice on such an effort (as in Homen's case). They were, however, present also during quiescence of the organ, since the lad's breathing even then remained interrupted and irregular. Muscular strength was somewhat weakened in the right arm (dynamometer, 32 l. 22 r.). Co-ordination, cutaneous sensibility, galvanic and faradic irritability

of muscles and nerves, trophic and vaso-motor functions, the higher senses and mind were quite normal. But cutaneous reflexes were greatly exaggerated, while tendon reflexes were considerably lowered (as in Homen's case). Under the influence of valerianate of zinc ($\frac{1}{3}$ grain, three times daily) and ascending galvanisation of the spine, considerable improvement steadily took place, and the contractions seemingly disappeared altogether.

VALERIUS IDELSON, M.D. (Berne).

END OF VOL. X.

LONDON: PRINTED BY WILLIAM CLOWES AND SONS, LIMITED, STAMFORD STREET
AND CHARING CROSS.